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WEIL'S DISEASE IN THE NORTH-EAST OF SCOTLAND

BY

J. SMITH

From the City Hospital Laboratory, Aberdeen

(RECEIVED FOR PUBLICATION JANUARY 7, 1949)

In 1939 Davidson and Smith reported on a series of 104 cases of Weil's disease which had occurred mainly amongst fish-workers in Aberdeen between the years 1934 to 1938. Since that time a further series of cases has been investigated and the present paper now deals with Weil's disease between the years 1934 and 1948, during which period 214 cases have been encountered. In the interval a number of papers dealing with various aspects of this infection in this and other countries have been published. Thus, Stuart (1939) and Jenkins and Sharp (1946) have reported cases amongst miners; Stewart and Witts (1944) and Gauld (1947) amongst bathers and military personnel; Broom and Alston (1948) have analysed a series of 195 cases which have been diagnosed in England, and in their list of occupations associated with the disease Army personnel take first place with 24 per cent., while farm, sewer, and fish workers account for 12, 11, and 7 per cent. respectively. Their overall fatality rate in one hundred cases in which they were able to obtain information was 19 per cent., while in 120 cases icterus was recorded in 107, or 90 per cent. In America the position has been reviewed by Meyer and others (1939), Stiles and Sawyer (1942), and Bertucci (1945); and in Holland a complete review has been published by van Thiel (1948). Information on the incidence of infection amongst rats with *L. icterohæmorrhagiæ* has been the subject of a note by Allcroft (1945).

TABLE 1

PERSONS EMPLOYED IN FISH-CURING PREMISES DECEMBER, 1948

No. of premises	No. of employees	Male	Female	Total
145	Under 10	296	358	654
62	11 to 25	367	535	902
13	26 to 50	180	269	449
8	Over 50	235	314	549
228	—	1,078	1,476	2,554

Epidemiology

The fishing industry in Aberdeen is mainly concerned with the preparation for the retail market of white fish which has been brought into port by trawlers from the North Sea and from the vicinity of Iceland. The work is undertaken by a large number of firms who may employ from two to over fifty workers. The findings obtained as a result of a recent census undertaken under the direction of the Chief Sanitary Inspector are given in Table 1. Thus, in a total of 228 premises 2,554 persons were employed, 1,078 being males and 1,476 females. These figures do not include the men employed in the transport section of the industry, as their numbers have not been recorded. The majority of the workers employed as filleters and freshers are in the age-group 15 to 25 years. A few older men are more frequently employed in the packing and transport sections of the industry. As the young women grow up many are married, and this produces a constant change of personnel from year to year. The actual number of firms and the number of employees have not varied much in recent years. When a survey was made some years ago (Smith and Davidson, 1936) it was shown that fifty-one or 24·2 per cent., of blood samples from 210 individuals gave a positive agglutination test against *L. icterohæmorrhagiæ* in dilutions varying from 1 in 30 to 1 in 1,000. Further, in a control series from 406 individuals with no connexion with the fishing industry, not a single positive reaction was obtained. From these and other observations it was considered that Weil's disease occurred in three grades of severity: (1) severe infections associated with jaundice, (2) mild infections with pyrexia and no jaundice, (3) latent or inapparent infections with no clinical manifestations. It is obvious, therefore, that in the following description of the disease as it appears in Aberdeen only cases belonging to categories 1 and 2 are included, and the many latent or inapparent infections are not recognized. The actual risk of contracting infection

TABLE 2

PLACE WHERE INFECTION WAS CONTRACTED

	Fish workers	Others	Total
Aberdeen	183	15	198
Aberdeenshire ..	0	8	8
Kincardineshire ..	1	6	7
Moray and Nairn ..	0	1	1
Banffshire	0	0	0
Orkney	0	0	0
Shetland	0	0	0
	184	30	214

must have been very great at one time, but this risk has apparently been considerably lessened in recent years judging by the reduction of the number of cases occurring annually.

The distribution of cases in the north-east of Scotland is given in Table 2, from which it is seen that out of a total of 214 no less than 198 occurred in the City of Aberdeen, eight in Aberdeenshire, seven in Kincardineshire, and one in Morayshire. In the Counties of Banff, Orkney, and Shetland, no case has so far been diagnosed despite the fact that sera sent in from cases of pyrexia of unknown origin, and jaundice, have often been tested for leptospiral agglutinins. It should be noted also that the medical practitioners in the area have shown an increasing awareness of the possibility of the occurrence of Weil's disease, and, further, the patients, particularly fish workers, often call the attention of the practitioner to the fact that they are possibly suffering from this disease. As regards the occupation of these patients, the details are given in Table 3, and the outstanding fact is that out of the total cases no less than 184, or 86 per cent., occurred amongst fish workers. It should be noted that the term "fish workers" includes individuals employed as carters who assist in the transport of fish from the market to the curing yards, and from the curing yards to the retail shops and railway station. These men are presumably infected while handling the slimy wooden boxes which have been contaminated by the excretions of rats. Workers in fish-and-chips shops are also included in this category, since they presumably get infected from boxes and their contents which were previously infected at the wholesale merchants' premises or in the actual shop of the retailer.

TABLE 3

OCCUPATIONAL INCIDENCE*

	First series : 1934–38	Second series : 1939–48	Total : 1934–48
Fish workers :			
filleters and freshers ..	93	77	170
porters and carters ..	5	7	12
fish-and-chips shop workers	—	2	2
Farm workers	2	11	13
Sewer workers	2	3	5
Workers in rag factory ..	1	1	2
Harbour immersion ..	1	1	2
Butcher and hide and tallow workers	—	2	2
Cook on cargo boat ..	—	1	1
Stablemen	—	1	1
Paper workers	—	2	2
Rat-catcher	—	1	1
Schoolboy	—	1	1
	104	110	214

* Fish workers 86 per cent. of total.

TABLE 4

AGE AND SEX INCIDENCES OF CASES

Age group	Sex	Cases ; first series : 1934–38	Cases ; second series : 1939–48	Total cases : 1934–48	Total deaths : 1934–48
Under 14	M	—	1	1	—
	F	—	—	—	—
14–20	M	9	19	28	1
	F	40	37	77	0
21–30	M	18	13	31	4
	F	10	6	16	0
31–40	M	8	18	26	4
	F	3	1	4	0
41–50	M	7	4	11	1
	F	0	2	2	0
51 +	M	8	8	16	9
	F	1	1	2	0
		104	110	214	19

Next in the list are farm workers with thirteen cases, and then sewer workers with five cases. It is obvious that by reason of the environmental conditions associated with their employment farm workers must be quite frequently exposed to the possibility of infection. The occurrence of five cases amongst sewer workers can be accounted for by the fact that large new construction projects have been under way in connexion with increased sewerage facilities. Two cases resulted from immersion in the harbour and one schoolboy admitted helping to clean ditches on the farm at home. The remaining eight cases had various occupations, and in each it is easy to envisage the conditions which would produce infection.

The age and sex distribution of the cases is given in Table 4, and the incidence as regards age and sex has been largely influenced by the preponderance of young people employed in the fishing industry.

TABLE 5

ANALYSIS OF DEATHS

	Cases	Deaths	% mortality
Total	214	19	8·9
Male	113	19	16·8
Female	101	0	0
Under 51 years { male ..	97	10	10·3
Under 51 years { female ..	99	0	0
51 years or over { male ..	16	9	56·2
51 years or over { female ..	2	0	0

Thus in the age group 14 to 20 years, out of 105 cases seventy-seven were females, while in the older age groups in which many of the female workers have given up their employment, the males are then in the majority and they carry out duties which would not at any rate be suitable for women. The youngest case in this series was a schoolboy aged 8 years, and it seems altogether very unusual to find so young a person infected. In all there were a total of 113 male to 101 female cases.

The case mortality rates are given in Table 5. Here the outstanding features are a low overall rate of 8·9 per cent. with a rate for males of 16·8 per cent. and none for females. Under 50 years of age the male mortality rate is 10·3 per cent., while over this age the mortality was nine deaths in sixteen cases, or 56·2 per cent. It seems noteworthy that despite there being 101 female cases there was not a single death, but it should be remembered that seventy-seven of these were under the age of 21 years. On the other hand had the mortality rate been similar for both sexes seven to eight deaths should have occurred in the female group.

Though the annual and monthly figures for all cases are comparatively small, Table 6 shows that up to 1941 the number of cases varied annually from seventeen to a maximum of twenty-six in 1938, while since 1941 the number has varied from four in 1944 to eleven in 1948. Taking the total monthly incidence the lowest figures were obtained in February and March, and the highest in September, there being a gradual rise and decline in the months preceding and following. It is difficult to be certain why there should be a maximum incidence in the early autumn, but it may have some relationship to the suitability of atmospheric temperatures for survival of the leptospira or it may be connected with biological changes in the rat population.

One factor which may account for the occurrence of Weil's disease in Aberdeen and its non-occurrence in other towns with a similar type of fishing industry (Hampson, 1946), is that the water supply in Aberdeen has an almost neutral *p*H, whereas in other towns the water may not have such a reaction.

TABLE 6

MONTHLY AND YEARLY DISTRIBUTION OF ALL CASES

19 :	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	Total
January ..	0	4	4	0	2	2	0	1	0	1	0	0	1	0	0	15
February ..	0	0	0	0	2	2	1	1	0	0	0	0	0	1	1	8
March	0	0	2	0	0	1	1	1	1	0	0	0	1	0	1	8
April	1	0	0	4	2	1	1	0	1	0	0	0	0	1	1	12
May	2	3	0	2	1	2	1	1	1	4	0	0	0	1	1	19
June	4	0	1	1	2	1	1	0	0	0	0	1	1	0	2	14
July	1	2	0	2	1	2	4	4	1	1	0	1	1	1	1	22
August ..	0	0	1	2	6	3	4	5	1	0	0	0	1	1	1	25
September ..	2	4	5	2	3	6	0	3	3	0	3	1	1	1	3	37
October ..	4	2	4	4	1	0	1	2	1	0	0	3	1	0	0	23
November ..	5	2	0	2	1	2	3	0	1	0	0	0	0	0	0	16
December ..	5	0	0	1	5	1	0	2	0	0	1	0	0	0	0	15
	24	17	17	20	26	23	17	20	10	6	4	6	7	6	11	214

It has been shown by Chang and others (1948) that leptospira may survive for four weeks in neutral tap water which has, in addition, a small amount of food supply. Further, since 1941 there has been a considerable decrease in the annual number of recognized cases of the disease and it is difficult to say why this should be so. In October 1940 a start was made to chlorinate the City water supply but during the last quarter of 1940 and the first half of 1941 chlorination was maintained for only about twelve hours per day. After June 20, 1941, chlorination became a more continuous process but the proper equipment for controlling this did not become available until the late autumn. It used to be the practice in the laboratory to prepare Vervoot's leptospiral medium with tap water, but after the introduction of chlorination the leptospira would not grow on media so prepared. As has already been shown by Smith and Davidson (1936), the presence of a chlorine derivative in water acts most successfully against leptospira. Chang and others (1948) have also shown that when residual chlorine was present to the extent of 1 part per million (p.p.m.) this organism only survived three minutes. At the present time only 0·5 p.p.m. are added to the municipal water supply after filtration, and by the time the water arrives at the tap no trace can be obtained of residual chlorine by the usual tests, although the water is practically sterile. It may be possible, therefore, that the chlorine combined with the organic matter present in the water is still to some extent inimical to the leptospira with which it comes in contact in the fish curing premises.

The incidence of the cases amongst fish workers as they occurred in certain curing yards is of some interest. Some curing yards have contributed quite a number of cases, the highest being seventeen, followed by fifteen in another and ten in a third. Obviously those premises in which these cases continue to occur require a thorough overhaul from the point of view of controlling rat infestation. As the fish curing yards are located in the vicinity of the harbour, and as there are frequently a number of business premises in each street, and as the premises in five streets have produced from eleven to twenty-three cases, this again is an obvious indication of the necessity to prosecute a vigorous campaign of rat destruction.

Clinical Manifestations

General.—These will be only briefly dealt with as they apply to the situation in Aberdeen and the north-east of Scotland. The course of the disease may be divided into three stages. The first or febrile stage lasts approximately one week, and during the first few days septicæmia seems to be the invariable rule, and only towards the end of the week do antibodies begin to appear in the serum. The initial symptoms of sudden onset, chill, fever, headache, and muscular pains, seem to be almost inevitable in every case. Prostration and vomiting are frequently present, and within a day or two conjunctivitis is evident in most. In fact the occupational history associated with the symptoms already described are almost indicative of Weil's disease as seen in Aberdeen. Jaundice appeared in 65 per cent. of the first series and 63 per cent. of the second series, or an average of 64 per cent. of all cases. Toxic nephritis, with albumin in the urine, is almost always present at some time during the first and later stages, and is often accompanied by the presence of red blood cells and casts.

The second or toxic stage is characterized by the absence of leptospira in the blood and the development of antibodies in the serum. In moderately severe cases the fever abates and the clinical condition improves ; but the jaundice continues and the toxic nephritis may be present for a month. In cases doing badly the jaundice becomes more marked, but this in itself need not cause alarm unless accompanied by severe hæmorrhage into the skin, mucous membranes, and various organs. The bad prognostic symptoms are reduced excretion of urine and a rapidly rising blood urea, death invariably being due to kidney failure which is accompanied by a falling blood pressure and a rapidly increasing pulse. The third or convalescent stage is characterized by the rapidly increasing titre of antibodies in the blood, the presence of leptospira in the urine, and the occurrence of secondary fever which is present in about half the cases.

In the previous series of 104 cases, six deaths were reported, but in the present additional 110 cases, thirteen have occurred, making nineteen in all. The particulars of the fatal cases are given in Table 7, from which it is seen that the shortest period of survival after the onset was recorded as five, and the longest twenty days, and since both these patients died at home the information on these points was obtained from the family doctor. The youngest fatal case was a man aged 18, and the oldest a man aged 72 years. The figures for icteric index and blood urea are dependent on the time after the onset at which the blood samples have been collected, but all fatal cases were clinically icteric.

Special Examinations

Blood Examination.—Blood examinations have been carried out on many cases and often there

has been little change from normal. Certain cases do show a definite polymorphonuclear leucocytosis of 10,000 to 30,000 leucocytes per c.mm. of blood, but the rise cannot be associated with any particular clinical symptom. In those cases showing severe toxæmia and purpura the loss of blood may even reduce the erythrocyte count to 1,500,000 and the hæmoglobin to 25 per cent.

Biochemical Examination.—The most important blood constituent from the point of view of prognosis is the urea. This was found to be over 40 mg. per 100 ml. of blood in 60 per cent. of cases in the first series, and 59 per cent. in the second series. As death is almost always due to uræmia, high values of 400 to 500 mg. per 100 ml. were encountered before a fatal termination. The icteric index was very variable in those cases showing jaundice but was frequently between 50 and 100. One case showed an icteric index of 200 but the severity of the jaundice is not such a bad prognostic sign as the severity of the toxic nephritis.

Meningitis.—Frequent mention of meningitis associated with a leptospiral infection occurs in the literature, and in the review by Walch-Sordrager (1939) an analysis of the incidence of this manifestation as it occurs in Holland is given. Of a total of 327 cases 12 per cent. showed evidence of meningitis, but in the present series there was a higher incidence amongst the non-jaundiced group, (23 per cent.) than amongst the jaundiced group, of whom only 5 per cent. were so affected. Many cases were lumbar punctured when the patient

TABLE 7

PARTICULARS OF FATAL CASES : ALL MALES

Case No.	Age	Days ill on admission	Blood				
			Bacteriological findings		Biochemical		
			Leptospira present	Serum agglutinins	Icteric index	Blood urea	Day of death
12	63	8	−	1/1000	—	—	9
39	45	8	−	1/300	60	397	9
43	61	6	−	1/30	89	250	8
79	68	Home	−	1/1000	63	89	8
81	59	7	+	1/10	67	161	10
99	27	4	+	— (1/300 later)	150	300	10
128	26	5	+	1/500	—	—	10
132	28	6	−	1/30	—	—	8
145	72	6	−	1/300	—	—	10
150	62	7	−	1/10	88	460	14
156	18	5	+	1/300	78	152+	11
177	40	4	+	—	180	320	9
187	54	4	+	1/1000	32	257	14
191	60	Home	+	—	—	—	5
194	62	Home	−	1/300	—	—	20
195	35	5	+	1/30	60	241	9
204	39	6	+	1/1000	90	220	9
205	33	4	+	1/30	100	315	8
208	25	5	+	1/300	58	526	11

complained of headache and slight neck stiffness but usually the protein and cell count of the cerebrospinal fluid was normal. Thus only 17 cases or 8 per cent. showed evidence of meningitis as judged by changes in the cerebrospinal fluid, and the details are given in Table 8. The protein content was usually from 50 to 80 mg. per 100 ml. with a maximum in one case of 120 mg. per 100 ml. The cell counts ranged from 6 to 600 per c.mm. of blood, and the relative proportion of polymorphs to lymphocytes also varied greatly. Repeated attempts by culture and guinea-pig inoculation have always failed to demonstrate leptospira in the cerebrospinal fluid.

Bacteriological Diagnosis

In the diagnosis of Weil's disease a proper application of bacteriological and serological tests is essential, and this again is dependent upon the correct understanding of the course of the infection. The disease is, during a short period, essentially a septicæmia, and the subsequent manifestations such as hæmorrhage, jaundice, and nephritis are a consequence of the toxæmia. The diagnosis at the septicæmic stage can only be made by culture of the blood in a suitable type of leptospiral culture media, or by inoculation of the specimen intraperitoneally into young guinea-pigs. Unfortunately the results of either procedure cannot become available for some days. The period required for leptospira to be demonstrable in a blood culture is variable and is at least a week, while inoculation of the blood clot into a guinea pig will produce a fatal result in seven to nine days if leptospira are present.

The recovery of the leptospira in this investigation has been, almost invariably, by guinea-pig inoculation, two animals being inoculated intraperitoneally

TABLE 8
CASES WITH MENINGITIS
FINDINGS IN CEREBROSPINAL FLUIDS

Case No.	Day of illness	Protein	Cells			
			Total	Polymorphs	Lymphocytes	Chlorides
30	6	60	238	67	33	706
	8	40	180	17	83	711
55	7	35	240	51	49	—
58	6	25	105	2	98	714
66	6	60	242	3	97	—
68	6	80	42	9	91	—
70	8	50	111	12	88	—
74	3	30	0	—	—	—
	5	50	30	58	42	—
75	7	65	243	32	68	—
91	9	65	84	26	74	—
102	11	70	20	3	97	—
106	6	120	78	36	64	710
113	8	35	93	9	91	708
128	9	45	37	17	83	714
168	7	70	6	0	6	696
182	9	50	75	32	68	730
186	6	80	600	20	80	710
194	4	80	370	69	31	724

TABLE 9

BLOOD : BACTERIOLOGICAL FINDINGS IN 187 CASES

Presence of leptospira as shown by guinea-pig inoculation

Day of disease	1	2	3	4	5	6	7	8	9	10
No. +ve	4	10	14	24	24	13	3	1	0	0
No. −ve	0	0	0	0	20	36	21	15	1	1
% +ve	100	100	100	100	55	27	12	6	0	0

through a large-bore needle with broken up clotted blood. This procedure is dispensed with when the patient has been ill for a period of seven or more days, and when the result of the serological test indicates a fairly high titre of antibodies. Table 9 summarizes the attempts at recovery of the organism. During the first four days after onset recovery of the leptospira is the rule, while on the fifth day only 55 per cent. of specimens showed the presence of the organism, and the percentage of positive results thereafter decreased still further. The results in Table 9 should be compared with summary of the serological tests in Table 10. The earliest day after the onset at which agglutinins have been encountered is the fourth, the number positive and the titre of the serum rapidly increasing in the succeeding days until by the ninth or tenth day with few exceptions a positive serological result is to be found. Two cases have been encountered in which, as noted by Broom (1948), the agglutinins did not appear until after the tenth day. This, however, is a very exceptional occurrence. Occasional difficulty in obtaining bacteriological proof also arises when a patient is admitted with a severe form of the disease about the sixth day after the onset and dies within a day or two ; by that time the leptospira may have disappeared from the blood and the antibodies may be absent or present only in low dilutions of the serum. Investigation of the urinary excretion of leptospira has not been carried out as a routine, as the results obtained are so variable and as the excretion occurs most frequently at a time when diagnosis has already been established by other means.

Treatment

Since the introduction of penicillin this antibiotic has been used in place of the specific antiserum in the treatment of cases of Weil's disease admitted to this hospital. There can be no doubt about the bactericidal and bacteriostatic action of penicillin on leptospira. The experimental work of Alston and Broom (1944), Heilman and Herrell (1944), and Larson and Griffiths (1945) on guinea-pig and hamster infections, has clearly shown the curative action of the drug when it is administered in the early stages of the infection. Clinical reports on treatment of human cases by Bulmer (1945) and Cross (1945) are now available, but the number of cases treated so far does not permit a proper evaluation of this form of therapy. It would seem, however, that penicillin must be administered within the first four days, when the septicæmic stage is present ; otherwise the action of the drug can avail the patient little when a toxic nephritis has developed. Since penicillin became available sixteen cases febrile on admission have so far been treated with doses ranging from 200,000 to 2,000,000 units daily, and four of these died. Sometimes it was thought that the penicillin had been responsible for the elimination of fever, but the patients were usually admitted at the time when defervescence would

TABLE 10

SEROLOGICAL TESTS ON SERUM

	Day of disease									
	1	2	3	4	5	6	7	8	9	10 and over
No. negative 1/10 dilution ..	2	7	9	17	24	20	7	2	0	2
No. positive 1/10 dilution ..	0	0	0	1	15	31	31	36	23	99
% positive 1/10 or greater ..	0	0	0	6	38	61	82	95	100	98

have occurred normally. So far as the highly toxic cases were concerned it did not seem to mitigate the course of the infection in any way. This series of cases is, however, too small to permit any general deduction to be made.

Workmen's Compensation

Weil's disease in the majority of instances occurs as an occupational hazard, and in Great Britain a legal precedent was established in the Court of Session, when an award was confirmed in the case of Raeburn *v.* Lochgelly Iron and Coal Company Ltd. (20 B.W.C.C. 637). Further, the Report of the Departmental Committee on Compensation for Industrial Disease, which was issued in 1936, recommended that infection by *L. icterohæmorrhagiæ* should entitle a workman to compensation for disablement, and that it should be scheduled as an industrial disease under Section 43 of the Workmen's Compensation Act, 1925. Actually there has been very little in the way of dispute about compensation. Men and women affected with the disease have, until the introduction of the National Insurance Act, received compensation automatically after a certificate had shown that they were undoubtedly suffering from a leptospiral infection. In some of the cases which ended fatally certain of the insurance companies have from time to time been inclined to dispute the fact that the disease was contracted by the individual while at work, but after an explanation was given of how the infection was contracted all cases have been settled out of court. With the introduction of the National Insurance Act the State has now taken over the responsibility for financial compensation from employers and insurance companies.

Summary

1. A survey has been made of 214 cases of Weil's disease which have occurred in the North-eastern area of Scotland between 1934 and 1948.

2. No fewer than 184 cases, or 86 per cent., occurred amongst fish workers.

3. The overall case mortality rate was 8·9 per cent., with a mortality rate for males of 16·8 per cent. and none for females.

4. Since penicillin became available sixteen cases febrile on admission have been treated with this antibiotic. It is believed, however, that antisera and antibiotics can influence the course of the disease only if given in the early stage of the septicæmia. It is not believed that their administration can influence the toxic nephritis which seems to be invariably fatal.

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VITAL CAPACITY MEASUREMENTS IN CORNISH TIN-MINERS

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Introduction

The first measurements of vital capacity were made by Hutchinson in 1846. During the 1914-18 war there was renewed interest in vital capacity measurement both in England and America following the suggestion that it might be used as an index of physical fitness. In an effort to find a method of calculating the normal figure for any individual, attempts were made on both sides of the Atlantic to relate the observed vital capacity of normal healthy males to some physical measurement, such as height, weight, surface area, stem height, or chest expansion.

The results of these investigations have been well summarized by Hewlett and Jackson (1922). These workers also calculated the correlation coefficients for vital capacity with height, height and weight, surface area (from du Bois' Tables) in a group of 400 male students aged 18 to 30 years. Lemon and Moersch (1924) compared the classifications and formulæ for determining the normal vital capacity proposed by Hutchinson (1846), Schuster (1911), Peabody and Wentworth (1917), Lundsgaard and van Slyke (1918), Dreyer (1919), West (1920), Hewlett and Jackson (1922), and Myers (1923). They concluded that West's formula was the most exact and the easiest to use. West stated that the vital capacity of males was 2·5 litres per sq. metre of surface area.

During the decade prior to the 1939-45 war there was a tendency not to relate the vital capacity to the physical measurements previously used, but to consider the lung volume subdivisions in relation to each other and to the total lung volume. At the same time these volumes were related to a new measurement, the so-called "radiological chest volume," a concept first introduced by Lundsgaard and van Slyke (1918) and developed by Hurtado and Fray (1933a) and Aslett, Hart, and McMichael (1939). This "radiological chest volume" is the theoretical thoracic volume. It consists in principle of the area of the lung fields and heart shadow (obtained by measurement of a chest film) multiplied by the antero-posterior diameter of the chest at full inspiration.

Hurtado and Fray, working on 50 healthy male medical students of 18 to 30 years, found a correlation between radiological chest volume and vital capacity of $+0{\cdot}7174 \pm 0{\cdot}0467$. The regression equation relating these variables was

$$\text{vital capacity} = (0{\cdot}24 \times \text{rad. chest vol. in litres}) + 1{\cdot}22 \text{ litres}$$

Aslett, Hart, and McMichael determined the relation of the lung volume and its subdivisions to various physical and radiological measurements in 64 South Wales coal-miners of 19 to 63 years with normal chest radiographs. They found that the correlation coefficient for radiological chest volume and vital capacity was $+0{\cdot}63$, and the regression equation was

$$\text{vital capacity} = (0{\cdot}0031 \times \text{rad. chest. vol. in c. ins.}) + 1{\cdot}25 \text{ litres}$$

As these authors point out, while Hurtado's films were taken at a distance of 6 ft., theirs were taken at $4\frac{1}{2}$ ft., which would give an increase in the area of the lung fields of approximately 4 per cent. over those of Hurtado. Also their measurements were made with the subject in a sitting posture while Hurtado's students were recumbent. This position would reduce the total lung volume by 9 or 10 per cent., the vital capacity by slightly less, and the radiological chest volume by 8 per cent. as compared with the sitting subjects (Hurtado and Fray, 1933b).

Parallel with these developments were attempts to estimate the normal reduction in the vital capacity with increasing age. Myers and Cady (1924) reviewed the literature and from their own observations clearly established this reduction. Kaltreider, Fray, and Hyde (1938) also summarized the results of previous work and estimated the changes with age in the lung volume and its subdivisions of a series of

normal males between 38 and 63 years. They found that in men of the fifth and sixth decades there were marked variations in the absolute values of the total lung volume and its subdivisions, but that the vital capacity might still be predicted with " a reasonable degree of accuracy " from the radiological chest volume. They concluded that the vital capacity decreases progressively from the third to the eighth decade, but a deviation of the observed volume of less than 20 per cent. from the calculated volume could be considered as within normal limits. A deviation of more than 20 per cent. would be due to other factors than age.

These authors and their co-workers also studied the relation of the observed lung volume and its sub-divisions to the corresponding volumes calculated from the radiological chest volume in 58 cases of pulmonary fibrosis. These were 57 men and one woman who, except for 4, had been exposed to inorganic industrial dusts from one to 40 years. Their ages ranged from 31 to 71 years (Hurtado and others, 1935). In a further paper Kaltreider, Fray, and Hyde (1937) correlated the observed measurements on the 58 cases already reported with the calculated volumes corrected for age as previously described (Kaltreider, Fray, and Hyde, 1938), and 32 new cases were added. The observations of these 90 cases were correlated with *x*-ray changes. The cases were divided into six groups depending on the degree of pulmonary fibrosis. Ignoring the two groups where the fibrosis was complicated by emphysema, a decrease in the mean observed vital capacity for each group, expressed as a percentage of the mean calculated volume for that group, was obtained with increasing degree of fibrosis.

Present Investigation

During the course of an investigation into pneumoconiosis among Cornish tin-miners conducted by the Department for Research in Industrial Medicine, measurements of vital capacity were made on the men examined clinically and radiologically at Geevor and South Crofty mines. The object was to determine the association between vital capacity and radiological lung changes and to assess the value of the vital capacity measurement as an index of a man's pulmonary condition during a survey lasting several years.

Initial Survey : March 1946

Methods.—A " British Benedict " basal metabolism apparatus was used with a recording drum operated by clockwork. The subject sat in an upright chair, in such a position that he could not see the recording drum. Each tracing lasted approximately four minutes. A recording of tidal air was made for the first 1½ to 2 minutes or until a steady base line was established, then one recording each of complemental air, reserve air, and vital capacity was taken with about ½ minute of tidal breathing after each. Various physical measurements were made on the majority of men examined clinically : weight (in singlet and trousers) to the nearest pound, standing height (in socks) to the nearest half inch, stem height and chest expansion both to the nearest quarter inch.

A detailed occupational history was taken from each man, from which could be calculated the length of exposure to dusts in metalliferous mining, and also the total exposure to silica-containing dusts incurred in a variety of additional occupations such as coal-mining, foundry work, and granite masonry. The man's age was also recorded.

The radiographs were taken with the Mobile Unit of the Joint Council of the British Red Cross Society and Order of St. John. The Mobile Unit is self-contained, with low tension generator of special design, having an output of 525 volts at a frequency of 100 cycles. Thermostatic control of the processing unit is provided in addition to air conditioning for interior working. Complete range of accessories is also housed in special fittings and containers. The *x*-ray unit is also of special design, 4-valve type with comprehensive control panel and accurate valve timer, reading 0·04–8 seconds. The technique used on the investigation was as follows :

K.V.P. 74,
M.A. 300,
DIST. 60 in.,
M.A.S. 15–25,
Focal Spot, 2 mm. rotating anode tube,
Exposure from ·06–·1,
Kodak Code 6 films,
Ilford Tungstate Screens.

Precise engine governing is incorporated in the 27-h.p. engine driving the alternator, maintaining the engine and frequency at constant speed, plus an efficient voltage regulation which provided for accurate control throughout the entire investigation.

Each chest radiograph was classified into one of four groups, I to IV, depending on the severity of lung changes, although for the following statistical analysis it was often necessary to combine groups II, III, and IV, owing to smallness of numbers. The films were classified as follows :

Group I : within the limits of normal.

Group II : excessive diffuse shadowing which might be described as doubtful reticulation.

Group III : reticulation.

Group IV : nodulation and/or massive shadows.

Complete data consisting of vital capacity, age, *x*-ray group, length of exposure in metalliferous mining and to siliceous dusts, were obtained for 134 men at Geevor ; a further 7 men were partially examined ; approximately 20 men refused to co-operate at all. At South Crofty, the above data was obtained for 128 men ; four men were partially

examined; again about 20 men refused to co-operate. Therefore a total of 262 complete observations was available for analysis.

Results.—The comparison of the mean and standard deviation of the vital capacity of workers at the two mines showed that for Group I (normal) and also for groups II, III, and IV combined (abnormal), the difference between the two mines was not significant (Table 1). The analysis of variance gives:

	Variance ratio	*P*
Normal *x*-ray group (I) ..	1·89	0·2 > P > 0·05
Abnormal *x*-ray group (II, III, IV)	0·51	P > 0·2
All *x*-ray groups	1·78	0·2 > P > 0·05

In each mine the mean vital capacity of the abnormal group was smaller than that of the normal group, and at Geevor this difference was significant.

	Variance ratio	*P*
Geevor	14·63	P < 0·001
South Crofty	3·15	0·2 > P > 0·05
Both mines combined	15·73	P < 0·001

However, vital capacity is negatively correlated with age. From the 262 observations of these Cornish data a significant correlation coefficient (r) between the two variables age and vital capacity was obtained: $r=-0{\cdot}453$, $t=8{\cdot}19$, and $P<0{\cdot}001$. A convariance analysis was therefore carried out to correct vital capacity for age differences between the normal and abnormal *x*-ray groups. As a result the reduction in the mean vital capacity of the abnormal compared with the normal group was no longer obtained. In other words the abnormal group consisted of older men, and the age factor masked any influence that the lung condition, as seen radiologically, might have exerted on the vital capacity.

But this method revealed differences between the two mines, regardless of *x*-ray classification. A significant reduction in the mean vital capacity of all South Crofty men compared with all Geevor miners was obtained: also the mean vital capacity for all normal men was significantly less at South Crofty than at Geevor. This is shown by the variance ratios.

	Variance ratio	*P*
Normal *x*-ray group ..	4·78	0·05 > P > 0·01
Abnormal *x*-ray group	0·36	P > 0·2
All *x*-ray groups ..	4·66	0·05 > P > 0·01

When all 262 observations were considered, a regression coefficient of 32·79 was obtained between vital capacity and age, and for vital capacity and length of exposure in metalliferous mining the regression coefficient was 0·85. The former was statistically significant (standard error=5·94, t=5·53 and $P<0{\cdot}001$) while the latter was not (standard error =6·51, t=0·13 and $0{\cdot}9>P>0{\cdot}8$). Therefore there is no advantage in estimating an "expected" vital capacity from length of exposure as well as from age, since the improvement to be obtained would be negligible.

In view of the significant difference between the mean vital capacity corrected for age of normal men at Geevor and South Crofty, it was felt that the vital capacity was associated with some other factor that differed between the two mines.

The men classified as normal, Group I, were therefore studied in more detail in an attempt to relate vital capacity to one of the many physical measurements taken. The data was complete for the 101 normal men at Geevor and for 74 of the 75 normal men at South Crofty. The description of these two populations in terms of the mean and standard deviations of the variables under consideration is summarized in Table 2.

It can be seen that at Geevor the mean figures were higher than at South Crofty for age, vital capacity, lengths of exposure to metalliferous mining and to silica dusts, and lower for height and weight, but none of these differences was statistically significant. At Geevor, however, the means for stem height and chest expansion were significantly higher than at South Crofty.

TABLE 1

VITAL CAPACITY OF MINERS BY *X*-RAY CATEGORY AND MINE

	X-ray group		
	Normal	Abnormal	Total
GEEVOR			
No. of observations ..	101	33	134
Mean vital capacity in c.cm.	3441	2859	3298
S.D. in c.cm.	786	668	797
SOUTH CROFTY			
No. of observations ..	75	53	128
Mean vital capacity in c.cm.	3275	2996	3159
S.D. in c.cm.	804	964	881
TOTAL			
No. of observations ..	176	86	262
Mean vital capacity in c.cm.	3370	2944	3230
S.D. in c.cm.	796	861	840

TABLE 2

THE MEAN AND STANDARD DEVIATION OF VITAL CAPACITY, AGE, LENGTH OF EXPOSURE TO METAL MINING, LENGTH OF EXPOSURE TO SILICA DUST, STANDING HEIGHT, WEIGHT, STEM HEIGHT, AND CHEST EXPANSION OF THE MINERS AT GEEVOR AND SOUTH CROFTY

Variable	Geevor		South Crofty		Total		Difference (Geevor–South Crofty)		
	Mean	S.D.	Mean	S.D.	Mean	S.D.	Difference	t	P
Number of observations ..	101		74		175				
Vital capacity in c.cm. ..	3441	786	3281	808	3373	797	160	1·32	0·2 > P > 0·1
Age in years..	33·65	10·35	30·70	9·73	32·41	10·17	2·95	1·91	0·1 > P > 0·05
Length of exposure in metal mines in years	9·69	7·91	7·84	7·44	8·91	7·75	1·85	1·57	0·2 > P > 0·1
Length of exposure to silica in years	12·52	8·90	10·45	8·18	11·65	9·22	2·07	1·45	0·2 > P > 0·1
Height in inches	66·35	2·71	66·43	2·70	66·38	2·70	−0·08	0·21	0·9 > P > 0·8
Weight in lb.	144·81	19·46	145·91	20·90	145·27	20·03	−1·10	0·36	0·8 > P > 0·7
Stem height in inches ..	35·00	1·24	33·69	1·31	34·44	1·42	1·31	6·72	P < 0·001
Chest expansion in inches ..	3·29	1·12	2·63	0·73	3·01	1·02	0·66	4·43	P < 0·001

Table 3 shows that the correlation between vital capacity and each of the variables age, length of exposure to silica dusts, height, and chest expansion was significant both at Geevor and at South Crofty. However, the differences between the corresponding correlations at the two mines were not significant. As these differences were not significant it was possible to pool the data and again a significant correlation was obtained between vital capacity and each of the variables.

The correlation between vital capacity and exposure in metalliferous mining was significant at Geevor but not at South Crofty ; for stem height and weight the position was reversed, being insignificant at Geevor and significant at South Crofty. The correlations for vital capacity with weight were not significantly different at the two mines ; the correlations for vital capacity with exposure in metalliferous mining and with stem height were significantly different at the two mines.

But none of this analysis accounts for the difference in the mean vital capacity of normal men at Geevor and South Crofty.

FOLLOW-UP SURVEY : 1947

Methods.—When the follow-up was conducted at Geevor Mine only, in 1947, it was decided to relate the vital capacity to the radiological chest volume.

The technique for the radiological examination was the same as that in 1946 but the films were taken on a 14 in. by 17 in. plate, instead of 12 in. by 15 in., to include the whole of the lung fields. Three plates each were taken for about one-third of the men *x*-rayed, and two plates each for another third.

The measurement of radiological chest volume was obtained from the area of the lung fields (including the heart shadow) on the *x*-ray film and from the antero-posterior diameter measured with callipers at the level of the nipple line at full inspiration. The boundaries of the lung fields as seen on the film were, for this purpose, assumed to be the line joining the inner edge of the crests of the first ribs, and laterally the line joining the inner crests of the ribs, and, at the base, the highest line of the diaphragm. This area was marked out on each radiograph with a yellow grease pencil (fig. 1) and measured with a planimeter ; the mean of five measurements of each plate was used. This area multiplied by the antero-posterior diameter at full inspiration gives the radiological chest volume.

As in 1946, the radiographs were classified into groups I to IV depending on the severity of lung changes.

The same respiratory apparatus was used as in 1946 with essentially the same circuit. The resistance of the circuit was reduced by the substitution of very light valves and by using less soda lime in the chimney : the reduction in the resistance was less fatiguing for the subjects but was not sufficient to result in a significant increase in the vital capacity measurement.* In the 1947 survey each recording consisted of a seven-minute tracing with five measurements of vital capacity, each

*Repeat tracings taken six months after the original measurements in 1946, after modification of the circuit, showed for 69 men at Geevor a mean increase in the vital capacity of 71·20 c.cm. S.D. 49·51, and for 59 men at South Crofty an increase of 120·76 c.cm. S.D. 564·85. Both these differences are statistically insignificant.

preceded by about one minute of tidal breathing. The temperature of the spirometer gases and the barometric pressure were noted at the end of each recording. The observed measurements of vital capacity were corrected to body temperature of 37° C. and pressure of 45 mm. Hg, not 47 mm. Hg as the air in the lungs is not fully saturated (Christie and Loomis, 1933).

The correction factor therefore is :

$$\frac{\text{B.P.} - (\text{V.P. at T})}{760 - 45} + \frac{273 + 37}{273 + \text{T}}$$

where B.P. = Barometric pressure in mm.Hg.
V.P. = Vapour pressure in mm. Hg.
T. = Observed temperature in °C.

TABLE 3

ZERO ORDER CORRELATION COEFFICIENTS (r) AND REGRESSION COEFFICIENTS (b) BETWEEN VITAL CAPACITY AND REMAINING VARIABLES

Correlation between vital capacity and :		Geevor	South Crofty	Total	Difference (Geevor–South Crofty)		
					Difference	t	P
Age	b	− 31·66	− 23·05	− 26·58	− 8·61	0·75	0·5>P>0·4
	r	− 0·42	− 0·28	− 0·34	− 0·14		
	t	4·56	2·45	4·74			
	P	P<0·001	0·05>P>0·02	P<0·001			
Length of exposure to metal mining ..	b	− 32·17	5·17	− 16·07	− 37·34	2·39	0·02>P>0·01
	r	− 0·33	0·05	− 0·16	− 0·38		
	t	3·41	0·40	2·08			
	P	P<0·001	0·7>P>0·5	0·5>P>0·2			
Length of exposure to silica	b	− 31·51	− 21·24	− 25·51	− 10·27	0·82	0·5>P>0·4
	r	− 0·36	− 0·25	− 0·30	− 0·11		
	t	3·80	2·21	4·06			
	P	P<0·001	0·05>P>0·02	P<0·001			
Height	b	105·73	167·02	131·02	− 61·29	1·52	0·2>P>0·1
	r	0·30	0·56	0·44	− 0·20		
	t	3·90	5·72	6·52			
	P	P<0·001	P<0·001	P<0·001			
Weight	b	7·79	17·50	12·11	− 9·71	1·69	0·1>P>0·05
	r	0·19	0·45	0·30	− 0·26		
	t	1·96	4·31	4·21			
	P	0·1>P>0·05	P<0·001	P<0·001			
Stem height	b	− 34·17	267·91	105·09	− 302·08	3·27	0·01>P>0·00
	r	− 0·05	0·43	0·19	− 0·48		
	t	0·54	4·07	2·51			
	P	0·5>P>0·4	P<0·001	0·02>P>0·01			
Chest expansion ..	b	314·51	509·78	349·03	− 195·27	1·50	0·2>P>0·1
	r	0·45	0·46	0·45	− 0·01		
	t	4·98	4·43	6·60			
	P	P<0·001	P<0·001	P<0·001			

The following information was therefor obtained about each subject: age, *x*-ray group, vital capacity (mean of five measurements) corrected for temperature and pressure, and radiological chest volume (mean of five measurements of each plate). These data were used in testing whether any relationship existed between vital capacity and radiological chest volume among the normal *x*-ray group, and if a correlation existed, whether these two variables could be used to distinguish cases on the borderlines between the four *x*-ray groups, and whether a man's progress could be assessed annually by expressing his observed vital capacity as a percentage of the normal "expected" volume calculated from the radiological chest volume.

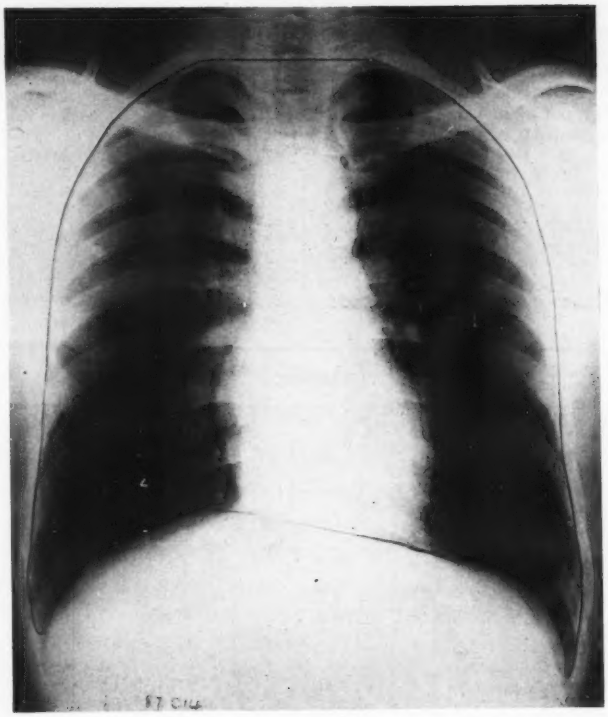

FIG. 1.—Radiograph showing area of lung fields marked out for measurement.

Results

Of the 134 men fully examined in 1946, 48 had left by 1947. Of the 86 remaining, 63 were fully and 11 partially examined, and 12 were absent or refused examination. In order to increase the numbers, additional men were examined for the first time, including new employees and men who had refused in 1946. Full data were therefore obtained for 105 men out of a total population of 168. Of the remainder, 30 were partially examined, and 33 were not examined through sickness, or through refusal to cooperate. Of these 33, 14 had been fully and 4 partially examined in 1946, leaving only 15 who had not been examined at all. Their details of age, occupation, and length of exposure at Geevor were obtained from the employers, and it is not thought that the exclusion of these men biases the results.

The inter-relation, between age, vital capacity, and radiological chest volume for 62 normal men is shown in Table 4. This shows that there was a significant and positive correlation between vital capacity and radiological chest volume of +0·63 (the same figure obtained by Aslett, Hart, and McMichael, 1939). The regression equation relating these two variables was:

vital capacity = (4·595 x rad. chest vol. in c. in.) − 134·20 c.cm.

A significant and negative correlation between vital capacity and age of −0·46 was also obtained. The radiological chest volume was unaffected by age.

When the analysis was extended to cover the four *x*-ray groups, that is, all 105 men examined in 1947 (Table 5), it was found that for radiological chest volume the variance ratio was less than 1 and therefore there was no association between radiological chest volume and *x*-ray categories. For vital capacity the variance ratio was 3·363 and $0{\cdot}05 > P > 0{\cdot}01$. Therefore the difference of vital capacity between *x*-ray groups was significant and there was a definite relationship between vital capacity and *x*-ray category.

TABLE 4

INTER-RELATION BETWEEN AGE, VITAL CAPACITY, AND RADIOLOGICAL CHEST VOLUME FOR 62 NORMAL MEN (GEEVOR, 1947)

	r	t	P
Vital capacity and age	−0·46	4·04	<0·001
Vital capacity and radiological chest volume ..	0·63	6·36	<0·001
Radiological chest volume and age	−0·07	0·57	$0{\cdot}6 > P > 0{\cdot}5$

TABLE 5

ANALYSIS OF VARIANCE OF RADIOLOGICAL CHEST VOLUME, AND OF VITAL CAPACITY WITH *x*-RAY CATEGORIES (105 OBSERVATIONS)

	Source of variation	Sum of squares	Degrees of freedom	Mean square
(*a*) Radiological chest volume..	Between *x*-ray groups	9695·65	3	3231·88
	Within *x*-ray groups	1907644·93	101	18887·57
(*b*) Vital capacity	Between *x*-ray groups	8126294·42	3	2708764·81
	Within *x*-ray groups	81358833·82	101	805533·01

However, vital capacity is negatively correlated with age, and there was a different age composition of the four *x*-ray groups. Therefore when the effect of age on the relation between vital capacity and *x*-ray groups was determined, it was found that the variance ratio was less than unity and that therefore vital capacity was not related to *x*-ray group when allowance was made for age (Table 6). It has already been shown that there was no relation between radiological chest volume and age, nor between radiological chest volume and *x*-ray group, and consequently the radiological chest volume was not related with *x*-ray category when allowance was made for age.

An alternative method of presenting the same data is shown in Table 7. The observed mean vital capacity decreased from group I to IV, but the mean age increased; that the decrease in mean vital capacity was a function of age is shown by the fact that the observed volumes for *x*-ray groups I, II, and III approximated very closely to the "expected" volumes calculated from the regression equation between age and vital capacity for the 62 normal subjects. In group IV the observed volume was lower than one would expect, even allowing for age, but the reduction was not statistically significant.

Table 7 also shows that the mean radiological chest volume was practically the same in all four *x*-ray groups. Consequently there was little difference between the "expected" mean vital capacity for each group, calculated from the regression equation between vital capacity and radiological chest volume obtained from 105 observations. As would be expected, similar but slightly higher volumes resulted when the regression equation obtained from the 62 normal observations only was used.

When the "expected" vital capacity was calculated in terms of the radiological chest volume, the overlap between the volumes for the four *x*-ray groups can be seen from fig. 2.

Discussion and Conclusions

The mean "expected" volumes for groups I, II, and III lie very close together, and the mean "expected" values for group IV are much lower, especially for the larger radiological chest volumes. When, however, the standard deviations of the mean values are considered, it can be seen that there is much overlapping between the "expected" volumes for each group.

Therefore when considering a subject whose *x*-ray appearance falls on the borderlines between *x*-ray groups, it is not possible to obtain assistance in classifying him by comparing his observed vital capacity with the calculated vital capacity using the regression equation between radiological chest volume and vital capacity.

Neither is it possible to trace a man's progress from year to year by relating his observed vital capacity to the "expected" vital capacity calculated in terms of radiological chest volume, as the limits within which the calculated "expected" vital capacity would fall are so wide as to include the whole possible range of observed vital capacity measurements. For example, it can be seen from the graph that the normal "expected" vital capacity for a man with a radiological chest volume of

TABLE 6

ANALYSIS OF VARIANCE OF RADIOLOGICAL CHEST VOLUME AND OF VITAL CAPACITY WITH *x*-RAY CATEGORIES CORRECTED FOR AGE

	Source of variation	Corrected mean square estimate
(*a*) Radiological chest volume	Between *x*-ray groups	2983
	Within *x*-ray groups	19058
(*b*) Vital capacity	Between *x*-ray groups	426506
	Within *x*-ray groups	673397

900 c.in. is 4000± 1300 c.cm. So that unless his observed vital capacity decreased to less than 2700 c.cm. it could not be classed as abnormal. If the observed vital capacity is expressed as a percentage of the " expected " volume without regard to the standard deviation of this volume, anomalous and misleading figures may result. For example, one man in group I had a vital capacity 64 per cent. of the "expected" volume, while another in group III was as high as 125 per cent., and one in group IV, 95 per cent.

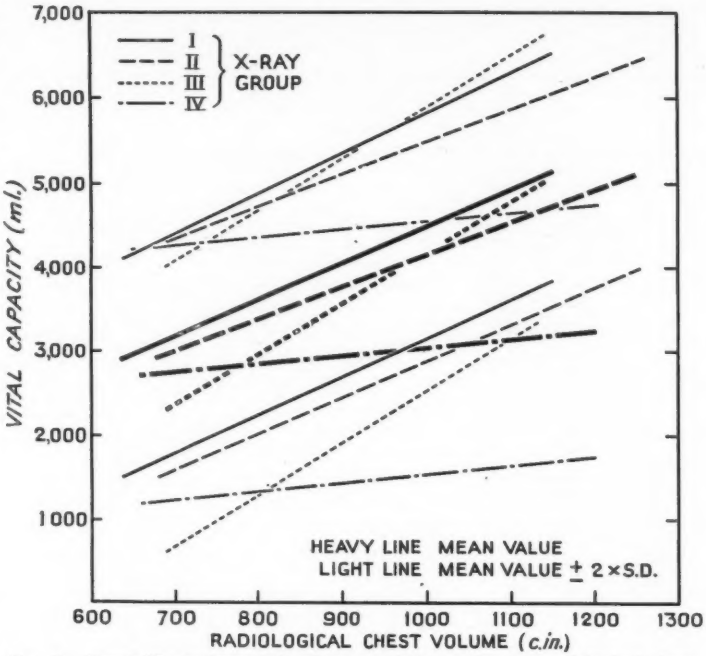

FIG. 2.—Graph illustrating relation between vital capacity and radiological chest volume for each *x*-ray group.

Summary

1. Since 1846 attempts have been made to relate the vital capacity of normal males to some physical or radiological measurement in order to calculate the " expected " vital capacity for any given individual. The literature is briefly outlined.

2. In the present investigation measurements of vital capacity were made on Cornish tin-miners examined clinically and radiologically at Geevor and South Crofty Mines, in order to determine the association between vital capacity and radiological lung changes, and to assess the value of the vital

TABLE 7

INTER-RELATION OF VITAL CAPACITY, RADIOLOGICAL CHEST VOLUME, AGE, AND *x*-RAY CATEGORY OF 105 OBSERVATIONS (GEEVOR, 1947)

	X-ray category			
	I	II	III	IV
Number of observations	62	22	15	6
Mean age in years	35·13	40·23	47·93	49·5
Mean radiological chest volume (R.C.V.) in sq. in. ..	889·56	872·34	874·09	907·73
Observed mean vital capacity (V.C.) in c.cm.	3953·5	3782·36	3417·41	2934·5
Calculated mean V.C. from age regression equation (62 normals only)	3953·5	3764·27	3478·24	3420·09
Calculated mean V.C. from R.C.V. regression equation (all 105 observations)	3801·81	3733·46	3740·41	3873·91
Calculated mean V.C. from R.C.V. regression equation (62 normals only)	3953·5	3874·36	3882·41	4036·98

capacity measurement as an index of a man's pulmonary condition during a survey lasting several years.

3. The initial survey in 1946 showed that at both mines the mean vital capacity of men with abnormal *x*-rays (groups II, III, and IV) was smaller than that of the normal men (group I) and at Geevor this difference was significant (variance ratio 14·63, $P<0·001$).

4. From all 262 observations combined, a significant correlation coefficient (r) was obtained between vital capacity and age: $r=0·453$, $t=8·19$ and $P<0·001$.

5. When allowance was made for age, the reduction in the mean vital capacity of abnormal men disappeared, that is, the age factor masked any influence that the lung changes, as seen radiologically, might have exerted on the vital capacity.

6. The normal men were studied in more detail and significant correlation coefficients were obtained at both mines between vital capacity and each of the variables age, height, chest expansion, and length of exposure to silica dusts. The correlation between vital capacity and the variables of stem height, weight, and exposure to metalliferous mining was significant in one or other of the mines.

7. In the 1947 follow-up survey at Geevor, the vital capacity was related to the radiological chest volume, which was found to be unaffected by age or radiological lung changes. A significant correlation coefficient between vital capacity and radiological chest volume was obtained for normal men: $r=+0·63$, $t=6·36$, $P<0·001$, and between vital capacity and age, $r=-0·46$, $t=4·04$, $P<0·001$.

8. The decrease in the observed vital capacity with increasing severity of lung changes was again shown to be a function of age.

9. When the "expected" vital capacity was calculated from the radiological chest volume, similar and overlapping values were obtained for each of the four *x*-ray groups. Therefore a comparison of the observed and "expected" vital capacities is of no diagnostic value.

10. The limits within which a man's "expected" vital capacity would fall are so wide as to include the whole possible range of his observed volume. Therefore this comparison cannot be used to trace a man's annual progress.

11. If the standard deviation of the "expected" vital capacity is ignored in such a comparison, anomalous and misleading figures may result.

I am greatly indebted to Dr. E. A. Cheeseman for carrying out the greater part of the statistical analysis and to Dr. W. J. Martin for its completion; without their continued co-operation and assistance this study would not have been possible. My thanks are also due to Dr. J. N. Agate for assistance and to Dr. J. Gilson for much helpful advice; to Dr. K. M. A. Perry for classifying the radiographs; to Mr. H. T. Ferrier, and to Mr. J. Maclagan for taking them; to Miss A. Huntley, and to Miss E. J. Fowler for technical assistance.

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SILICOSIS IN THE POTTERIES

SOME OBSERVATIONS BASED ON SEVEN HUNDRED AND FIFTY NECROPSIES

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Manufacture of pottery in Great Britain is largely concentrated in North Staffordshire, where it constitutes the staple industry of the district, known throughout the world as the "Potteries." According to the latest returns of the Ministry of Labour the chief sections of the industry and the respective number of workmen employed, which may be considered representative of the industry in the present century, are as shown in Table 1.

TABLE 1

NORTH STAFFORDSHIRE POTTERY INDUSTRY: PERSONS EMPLOYED IN VARIOUS SECTIONS

General earthenware	25,387
China	7,200
China and earthenware (mixed)	5,053
Sanitary earthenware	3,062
Earthenware tiles	6,804
Electrical porcelain	5,563
Jet and Rockingham	1,753
All sections	54,822

Women and girls constitute 55 per cent. of all operatives, but they are mainly employed in the manufacture of tiles and in decorating processes.

Potters' rot or potters' asthma, silicosis or silicosis accompanied by tuberculosis, is a well-recognized risk of work in the industry, and each year causes a considerable number of deaths (Annual Reports of H.M. Chief Inspector of Factories).

Over the last seventeen years this represents an annual incidence of 44 deaths, with an average age at death of 59·1 years (Table 2).

During the years 1931–1946 the Stoke-on-Trent panel of the Medical Board for Silicosis, in pursuance of their statutory duties, attended 750 necropsies in local pottery workers. In each case the death of the workman had been reported to H.M. Coroner by a medical practitioner, a Registrar of Deaths, a relative, or a trade union official, so that inquiry might be made into the cause of death, with particular reference to silicosis. The primary object of the procedure was in relation to claims for workmen's compensation. In practice this meant that almost every death occurring in potters exposed to the risk of silicosis was investigated by post-mortem examination. For various reasons the Medical Board were unable to be present at every necropsy, but attendance was made at 90 per cent. of the total cases. Only those actually attended are included in this paper, otherwise there has been no selection. The series probably represents the largest group which has come under the notice of one observer in any single industry or process in Great Britain.

TABLE 2

SILICOSIS OR SILICOSIS ACCOMPANIED BY TUBERCULOSIS: FATAL CASES
(Great Britain)

Year	No. of deaths	Average age at death (in years)
1932	53	56·6
1933	53	57·6
1934	43	58·1
1935	54	57·2
1936	42	57·3
1937	63	58·5
1938	52	58·1
1939	46	59·4
1940	48	60·4
1941	41	59·3
1942	40	59·7
1943	32	60·3
1944	30	55·4
1945	32	62·9
1946	35	62·2
1947	43	61·5
1948	45	62·5

The cause of death as certified after full pathological investigation may be summarized as in Table 3. No death occurred in the manufacture of Jet and Rockingham ware, that is the manufacture of brown teapots from local marls; but, as shown below, they were distributed over all other sections.

From these figures it is not possible to compute comparative mortality rates for the separate groups; this has been dealt with by Middleton (1936). It should be noted that the disease occurs in women; clinical experience suggests that the sexes react alike to the risk.

Note on Cases in China (Clay Processes).—English bone china is composed of Cornish stone, china clay, and calcined bone, but unlike earthenware contains no added flint or free silica. Accordingly it has always been difficult to account for cases of silicosis in the clay processes of china manufacture, especially as these workmen are highly skilled and there is little or no interchange of labour between the china and earthenware sections of the industry, or between flint and clay processes. To some extent powdered flint, before alumina was substituted for it (Plant, 1939), was used as a parting powder on the boards on which the ware is carried; and there was universal contamination of all working places by flint from the placing shops, the ovens, and the china biscuit warehouse. Indeed, it was a well-known fact that powdered flint was dispersed into the streets adjoining china manufactories. These are relevant facts, but it is not claimed that they contain the whole explanation for the occurrence of silicosis in this branch of the industry.

TABLE 3

CERTIFIED CAUSE OF DEATH

Cause of death	No. of cases	%
Silicosis	217	28·9
Silicosis accompanied by tuberculosis	241	32·1
Other causes (silicosis present) ..	142	19·0
Other causes (no silicosis) ..	150	20·0
All causes	750	100·0

Certified Cause of Death

(Group Analysis and Commentary)

No Silicosis.—In 150 cases there was no evidence of silicosis even after histological investigation by Dr. S. Roodhouse Gloyne (Tables 4 and 5).

The predominance of respiratory diseases is of no statistical significance, as the series did not include all, or even a random sample, of deaths among potters. There was selection in that only such cases as might reveal silicosis were reported to the

TABLE 4

CAUSE OF DEATH ACCORDING TO OCCUPATION OR PROCESS

Occupation or process	Cause of death				All causes
	Silicosis	Silicosis + tuberculosis	Other causes (silicosis present)	Other causes (no silicosis)	
China-flint processes	24 (6)	32 (8)	14	11 (1)	81 (15)
China-clay processes	8 (1)	12 (1)	6	13 (1)	39 (3)
Polisher	12	15	2	6	35
Millman	21	23	10	18	72
Earthenware biscuit placer	7	2	10	28	47
General earthenware-clay processes..	97 (5)	113 (15)	80 (4)	59 (17)	349 (41)
Sanitary earthenware	22	28	19	7	76
Earthenware tiles	22 (4)	14 (10)	1	2 (1)	39 (15)
Electrical porcelain	4	2	—	6	12
All	217 (16)	241 (34)	142 (4)	150 (20)	750 (74)

The bracketed numbers, in this and other tables, indicate females and are included in the totals.

coroner. For the same reason it is not permissible to argue that the high proportion of deaths from pulmonary tuberculosis suggests that this condition may be related to the inhalation of silica even in the absence of manifest silicosis. Chemical analysis of the ash of the whole or a part of a lung and micro-incineration of sections were for a time undertaken but were abandoned as of no practical value.

Eleven deaths were attributed to primary bronchial carcinoma; full reference will be made to this subject later.

The age at death (Table 6) reveals a point of interest. The potter is a highly skilled craftsman, entering his trade on leaving school and continuing it throughout his working life. As will appear later in Tables 7, 10, and 13, the disease, silicosis or silicosis accompanied by tuberculosis, is frequently manifest after the age of forty years, yet, as Table 6 shows, a workman may spend a whole lifetime in dangerous processes without contracting the disease. This conforms to experience in other silicosis-producing industries and processes. The disease involves the problem of soil as well as seed.

Silicosis Accompanied by Tuberculosis.—In this context tuberculosis means active tuberculosis of the lungs (T.B. plus), and in 241 cases the combined disease was the cause of death. The distribution of these by age group and occupation is set out in Table 7.

The relatively high incidence of fatal tuberculosis in late middle life is apparent which accords with

TABLE 5

CAUSE OF DEATH IN 150 CASES IN WHICH THERE WAS NO EVIDENCE OF SILICOSIS

Cause of death	Men	Women	Persons
Tuberculosis of lungs ..	34	13	47
Primary bronchial carcinoma	10	1	11
Other respiratory diseases	34	1	35
Influenza	4	—	4
Cardio-vascular diseases	29	3	32
Renal diseases	4	1	5
Malignant diseases: other organs	6	—	6
Miscellaneous	9	1	10
All diseases	130	20	150

TABLE 6

AGE AT DEATH IN RELATION TO OCCUPATION OR PROCESS (150 CASES)

Age group	Occupation or process									All
	China (flint)	China (clay)	Polisher	Mill-man	Earth. bisc. placer	Gen. earth. (clay)	San. earth. (clay)	Earth. tiles	Elect. porc.	
Under 20 ..	—	—	—	—	1	2 (2)	—	—	—	3 (2)
20–24	—	—	1	—	1	2 (1)	—	1 (1)	—	5 (2)
25–29	—	—	1	—	1	3 (2)	—	—	—	5 (2)
30–34	1	1	1	—	2	3 (3)	—	—	—	8 (3)
35–39	—	1	—	2	1	3 (3)	—	—	—	7 (3)
40–44	2	3 (1)	—	1	2	5 (3)	1	—	3	17 (4)
45–49	3	2	—	1	4	7 (1)	4	—	—	21 (1)
50–54	—	—	3	6	8	13 (1)	—	—	—	30 (1)
55–59	4 (1)	2	—	5	3	7	1	—	1	23 (1)
60–64	—	2	—	2	3	6	—	—	—	13
65–69	—	1	—	1	2	4	1	—	2	11
70+	1	1	—	—	—	4 (1)	—	1	—	7 (1)
All ages ..	11 (1)	13 (1)	6	18	28	59 (17)	7	2 (1)	6	150 (20)

the original observations of Brownlee (1920) on the epidemiology of phthisis in Great Britain among workers in silica.

Reference to Table 3 reveals that there was evidence of silicosis in some degree in 600 cases, and in 241 this was combined with active tuberculosis of the lungs. Tuberculosis is recognized as the most common and fatal complication of silicosis, but this incidence (40 per cent.) is considerably below what is usually accepted. The explanation of this is not easy but there are several possibilities. It may be that some authors have computed the incidence of complicating tuberculosis as a percentage of the deaths actually attributed to silicosis or silicosis accompanied by tuberculosis, thereby omitting those cases in which silicosis was present but in which death was due to " other causes." In this instance 241 cases would thereby be related to 458 cases (silicosis 217, silicosis accompanied by tuberculosis 241), which represents 53 per cent.

On the other hand the estimates may have been based on the clinical diagnosis of the disease, in which tuberculosis or phthisis did not necessarily involve tubercle bacilli in the sputum.

As will appear later (Table 11), in 178 out of 217 cases of silicosis the disease was of the confluent or massive type, a lesion which has been variously described as infective silicosis or silico-tuberculosis. In the overwhelming majority of these, wasting and general physical deterioration were characteristic, so that on clinical grounds, in the absence of post-mortem examination, death might quite rightly be attributed to what the older physicians designated " fibroid phthisis." From this it would follow that 419 of 600 cases, or on the other basis specified, 419 of 458 cases, representing respectively 70 and 91 per cent., were due to fibroid phthisis, though this is not synonymous with silicosis accompanied by active tuberculosis.

There is another fact which may be relevant, namely that nowadays silicosis in potters is, with rare exceptions, a disease of late middle or advanced age, just when death from acute intercurrent or degenerative diseases is most frequent. In such circumstances the disease may not be permitted to pursue its natural course because it is prematurely interrupted by the extraneous condition. In 1892 Arlidge (cited by Collis, 1919) recorded that, " The mean age at death of male potters, aged twenty and upwards, was forty-six and a half years, while that of non-potters stood at fifty-four." Now by contrast the average age at death from silicosis or silicosis accompanied by tuberculosis among potters is fifty-nine years (Table 2). This association of tuberculous infection and pneumoconiosis, the problem of soil and seed, is still the outstanding unresolved scientific problem in this field. The preceding discussion emphasizes the need for precise definition of the methods used in compiling statistical data bearing on this aspect of the problem.

An interesting subject for consideration is the degree of silicosis associated with fatal tuberculosis of the lungs, especially as it involves the legal significance of ' silicosis accompanied by tuberculosis ' under Workmen's Compensation or, as it now is, under the National Insurance (Industrial Injuries) Act, 1946. To examine this in the present group, silicosis has been divided into macroscopic nodular silicosis, confluent or massive silicosis, and silicosis which is not obvious to the naked eye but can be proved by microscopic examination (Table 8).

The conclusion is that tuberculosis may become manifest and may progress to a fatal termination even when the degree of silicosis is minimal, and, what is very important, when it is unlikely to be detectable on radiographic examination of the lungs. There is the further practical consideration that every case of silicosis that has reached a diagnosable stage is potentially a case of active tuberculosis, involving danger to contacts in the home and at work. All certified cases of silicosis should be under periodical medical supervision at a chest clinic.

In those cases in which gross tuberculosis is associated with microscopic or even slight nodular silicosis, it is frequently asserted that the silicosis is merely a coincidental lesion and that the two conditions are not causally related. Furthermore the question is often raised which condition came first. Such nice discrimination is not possible and so far as the legal problem, in respect of Workmen's Compensation, is concerned, the statute has recognized the difficulties and resolved them in the designation, " silicosis *accompanied* by tuberculosis " ; there is no question either of degree or of priority.

During life the physician is frequently confronted with a patient who has been exposed to a recognized silica hazard and in whom the clinical and radiographic picture is one of extensive tuberculosis of the lungs. In such a case it is fatuous to imagine that it is possible to distinguish radiographically between silicotic and tuberculous opacities and it is extremely doubtful whether the age of the patient affords any clue. As questions of Workmen's Compensation or Industrial Injury are involved, the diagnosis must differentiate between simple tuberculosis and silicosis accompanied by tuberculosis. All that is possible is a presumptive diagnosis based almost entirely on a critical assessment of the risk as revealed by a precise detailed occupational history. By this means experts

TABLE 7

AGE AT DEATH IN RELATION TO OCCUPATION OR PROCESS: SILICOSIS ACCOMPANIED BY TUBERCULOSIS

Age group	Occupation or process									All
	China (flint)	China (clay)	Polisher	Mill-man	Earth. bisc. placer	Gen. earth. (clay)	San. earth. (clay)	Earth. tiles	Elect. porc.	
Under 20 ..	—	—	—	—	—	—	—	—	—	—
20–24	—	—	—	—	—	—	—	—	—	—
25–29	—	—	—	—	—	—	—	1 (1)	—	1 (1)
30–34	—	—	—	—	—	—	1	2 (1)	—	3 (1)
35–39	1	—	1	—	—	3 (1)	1	2 (2)	—	8 (3)
40–44	1	1	5	5	—	12 (5)	1	4 (2)	—	29 (7)
45–49	3 (3)	1	2	3	—	12 (5)	3	3 (3)	—	27 (11)
50–54	10 (4)	1	2	5	—	23	6	1 (1)	—	48 (5)
55–59	8 (1)	1	1	4	—	21 (2)	—	—	1	36 (3)
60–64	5	3 (1)	2	5	1	16 (2)	7	—	—	39 (3)
65–69	2	3	2	1	1	22	4	1	—	36
70+	2	2	—	—	—	4	5	—	1	14
All ages ..	32 (8)	12 (1)	15	23	2	113 (15)	28	14 (10)	2	241 (34)

TABLE 8

DEGREE OF SILICOSIS ASSOCIATED WITH FATAL TUBERCULOSIS

Occupation or process	Degree of silicosis			Total
	Microscopic	Nodular	Confluent	
China (flint)	6 (3)	7 (2)	19 (3)	32 (8)
China (clay)	5 (1)	2	5	12 (1)
Polisher	1	9	5	15
Millman	3	8	12	23
Earth. biscuit placer	—	—	2	2
General earth. (clay)	25 (7)	37 (4)	51 (4)	113 (15)
Sanitary earth.	5	11	12	28
Earth. tiles	1 (1)	5 (3)	8 (6)	14 (10)
Elect. porcelain	—	2	—	2
All	46 (12)	81 (9)	114 (13)	241 (34)

achieve almost complete accuracy, as may be proved later by post-mortem examination of patients who have died.

Chronic ulcerative tuberculosis is not uncommon in silicotic cases, but a terminal acute disseminated tuberculosis of the lungs, having its origin in a small localized cavity, is a fairly frequent finding. Tuberculous pneumonia also occurs, and cannot always be distinguished by naked eye examination from non-tuberculous varieties. This also applies to pericarditis in these cases. On several occasions tubercles and tubercle bacilli were demonstrated in the heart muscle. These observations show how important it is that the histological examination should always include sections suitably stained for tubercle bacilli. If the presence or absence of silicosis in cases of gross tuberculosis is to be determined, much depends on the selection of suitable histological material. The hilum glands should always be examined, together with blocks of tissue from the apparently healthy part of the lung. As far as possible these should include the pleura.

Silicosis accompanied by tuberculosis is certainly more rapidly fatal than simple silicosis, but this does not signify that tuberculosis associated with silicosis is a more fulminating type than simple tuberculosis, as is suggested by Collis (1919), Nicholson (1923), Thompson and Ford (1927), and Heffernan (1926). Beattie (1916) submits the contrary, namely, that the silicotic fibrosis may actually hinder the progress of the associated tuberculosis. The present series did not reveal any difference, which accords with our experience in a group of 310 Sheffield metal and cutlery grinders who were specifically investigated with this problem in view (Meiklejohn, 1929).

TABLE 9

FATAL HÆMOPTYSIS IN TWENTY-THREE CASES

Disease	No. of cases	Fatal hæmoptysis
Simple tuberculosis	47 (13)	5 (1)
Silicosis + tuberculosis ..	241 (34)	18 (5)

The terminal event in 23 cases was fatal hæmoptysis (Table 9).

Silicosis without Tuberculosis.—There remain for consideration 359 cases (Table 3) in which silicosis

TABLE 10

DEATH DUE TO SILICOSIS: AGE AT DEATH IN RELATION TO OCCUPATION OR PROCESS

Age group	Occupation or process									All
	China (flint)	China (clay	Polisher	Millman	Earth. bisc. placer	Gen. earth. (clay)	San. earth. (clay)	Earth. tiles	Elect. porc.	
Under 20 ..	—	—	—	—	—	—	—	—	—	—
20–24	—	—	—	—	—	—	—	—	—	—
25–29	—	—	—	—	—	—	—	—	—	—
30–34	—	—	—	—	—	—	—	2 (2)	—	2 (2)
35–39	—	—	—	—	—	—	—	—	—	—
40–44	1 (1)	—	1	2	—	1	—	3 (1)	1	9 (2)
45–49	1	—	1	3	—	1	—	—	1	7
50–54	5 (1)	1	3	1	1	11 (1)	1	6	—	29 (2)
55–59	7 (3)	1	4	4	2	18 (1)	5	2 (1)	1	44 (5)
60–64	8 (1)	1	1	6	3	24 (3)	8	4	1	56 (4)
65–69	2	3	2	3	—	29	7	5	—	51
70+	—	2 (1)	—	2	1	13	1	—	—	19 (1)
All ages ..	24 (6)	8 (1)	12	21	7	97 (5)	22	22 (4)	4	217 (16)

TABLE 11

DEATH DUE TO SILICOSIS: MODE OF DEATH AND RELATED DEGREE OF SILICOSIS

Terminal event	Degree of silicosis			Total
	Microscopic	Nodular	Confluent	
Inanition	—	2	46 (3)	48 (3)
Heart failure: sudden	—	1	12 (1)	13 (1)
congestive	2	14	52 (2)	68 (2)
progressive	—	2	14 (3)	16 (3)
Pericarditis	1	—	1	2
Syphilitic mesaortitis	—	—	3	3
Pneumonia	—	15 (1)	31 (3)	46 (4)
Bronchiectasis	—	—	1	1
Spontaneous pneumothorax	—	—	4 (1)	4 (1)
Influenza	—	1	9 (2)	10 (2)
Nephritis	—	1	1	2
Miscellaneous	—	—	4	4
All	3	36 (1)	178 (15)	217 (16)

TABLE 12

DEATH DUE TO "OTHER CAUSES" (SILICOSIS PRESENT): MODE OF DEATH AND RELATED DEGREE OF SILICOSIS

Terminal event	Degree of silicosis			Total
	Microscopic	Nodular	Confluent	
Heart failure: sudden	12	1	—	13
Cardio-vascular degeneration	18	4	—	22
Syphilitic mesaortitis	2	2	—	4
Pneumonia	17 (1)	—	—	17 (1)
Bronchiectasis	2	—	—	2
Primary bronchial carcinoma	7	1	5	13
Influenza	2	—	—	2
Nephritis	9	3	3 (1)	15 (1)
Cystitis and hydronephrosis	1	1	—	2
Cerebral hæmorrhage and thrombosis	2	3	2	7
Malignant disease: various organs	7 (1)	8	5	20 (1)
Miscellaneous diseases	12 (1)	7	6	25 (1)
All	91 (3)	30	21 (1)	142 (4)

TABLE 13

DEATH DUE TO OTHER CAUSES " " (SILICOSIS PRESENT) : AGE AT DEATH IN RELATION TO OCCUPATION OR PROCESS

Age group	Occupation or process									Total
	China (flint)	China (clay)	Polisher	Mill-man	Earth. bisc. placer	Gen. earth. (clay)	San. earth. (clay)	Earth. tiles	Elec. porc.	
Under 20 ..	—	—	—	—	—	—	—	—	—	—
20–24	—	—	—	—	—	—	—	—	—	—
25–29	—	—	—	—	—	—	—	—	—	—
30–34	—	—	—	—	—	—	—	—	—	—
35–39	—	—	—	—	—	—	—	—	—	—
40–44	—	—	1	1	1	3 (1)	—	—	—	6 (1)
45–49	3	—	1	—	—	5 (1)	2	—	—	11 (1)
50–54	—	—	—	1	2	14	—	—	—	17
55–59	6	—	—	—	1	10	4	1	—	22
60–64	4	2	—	4	2	21 (1)	5	—	—	38 (1)
65–69	1	2	—	1	1	15 (1)	4	—	—	24 (1)
70+	—	2	—	3	3	12	4	—	—	24
All ages ..	14	6	2	10	10	80 (4)	19	1	—	142 (4)

was present without any evidence, macroscopic or microscopic, of active tuberculosis. In 217 of these (Table 10) silicosis was adjudged the primary cause of death, though, as will appear in Table 11, the terminal event or illness assumed a variety of forms.

Death in the remaining 142 cases was attributed to " other causes," such silicosis as existed being considered unrelated and non-contributory (Tables 12 and 13).

The "miscellaneous" group includes pernicious anæmia, myelogenous leukæmia, gangrene of the foot, acute transverse myelitis, malignant endocarditis, extra-dural hæmorrhage, acute alcoholic poisoning, acute appendicitis, acute hæmorrhagic pancreatitis, cirrhosis of the liver, cholæmia (gallstones), intestinal obstruction, and rupture of aortic aneurysm.

Certain matters merit brief comment.

Inanition.—In cases of massive silicosis of long-standing this is a fairly common terminal event. Through lack of sleep, loss of appetite, and the physical exhaustion associated with harassing cough and distressing dyspnœa, the patient progressively deteriorates; life just fades out. The condition is an entity and should not be classified as cardiac failure.

The Heart and Heart Failure.—The heart and mediastinum are seldom obviously displaced or distorted, this probably being due to firm fixation by the generalized intra-thoracic adhesions that are so characteristic of silicosis. In the clinical assessment of cases the extent and significance of these adhesions are frequently overlooked, a fact that emphasizes the need for fluoroscopic examination as a preliminary to radiography.

The size of the heart in silicosis shows no constant alteration and is not directly related to the degree of lung fibrosis. In the most advanced stages of massive consolidation, where the disease is known to have existed for many years, the heart may be of normal size. Whenever possible the organ was weighed, and while there was a tendency to a slight increase, this seldom exceeded a few ounces except in cases of concomitant disease. The increase is due to hypertrophy of the muscle of the right ventricle and is particularly obvious in cases with emphysema. Gross enlargement with increase of weight is usually associated with hyperpiesia, syphilitic aortitis, chronic endocarditis, or renal disease. The significance of these observations is that cardiac enlargement is seldom to be detected clinically or radiographically in cases of silicosis and when gross

enlargement does appear it is usually due to some other disease.

The classification of heart failure into sudden, congestive, and progressive may appear unnecessarily precise, but it does serve to reflect three well-defined clinical pictures of the disease in the terminal phase and is not without value in prognosis. In sudden death the workman, apparently in his usual health, expires more or less instantaneously and at necropsy the characteristic feature is considerable dilatation of the right ventricle. Cases in which definite coronary occlusion was established as the cause of the event are not included but appear as deaths due to "other causes (silicosis present)." Congestive heart failure denotes those patients who during the last few weeks or months become increasingly dyspnœic, with congestion and cyanosis of the face and extremities, but in whom dropsy is not present. They manage to keep walking about at home and they are confined to bed only during the last few days. At necropsy the right ventricle and auricle are considerably dilated and distended with blood clot. Finally there are those patients who develop œdema, which, commencing round the ankles, gradually extends and is followed by anasarca, ascites, and even hydrothorax. This is usually a long illness during which the patient is bedridden. Such cases are uncommon nowadays.

Attention was directed to the occurrence of sclerosis of the pulmonary vessels at the hilum, but of this there was no evidence. Extensive atheromatous degeneration of the abdominal aorta and its branches was common, even when the thoracic aorta was comparatively healthy.

Chronic Nephritis.—Collis and Yule (1933) have asserted that in their opinion renal disease is more common in silicotics than among the general population of corresponding age. This is not supported by the present survey and does not accord with clinical experience.

The Lungs and Pleura.—Attention has already been directed to the occurrence of adhesions. These are usually sessile and tough, and often result in complete obliterative pleurisy. Over the apices, particularly in cases of massive fibrosis, the pleura may be one quarter of an inch in thickness and may resemble cartilage. The attachment of the lung to the chest wall is particularly noticeable over the apices posteriorly, which helps to explain the clinical observation of marked impairment of percussion in these zones posteriorly while resonance anteriorly over the clavicles persists. Having regard to the extent and density of the adhesions it is interesting to note how rarely the intra-thoracic nerves are involved. In clinical practice I recall only two cases of laryngeal and one of diaphragmatic paralysis. Similarly traction diverticula of the œsophagus are seldom revealed at necropsy, though this does not mean they may not be demonstrated more often radiographically during life.

Emphysema, a frequent complication, is of the bullous type in which the bullæ appear as bunches of grapes of varying size along the free margins of the lungs, over the bases, and occupying the costo-diaphragmatic sulci. Bullæ as large as tangerine oranges are not uncommon. Rupture of a bulla results in pneumothorax, but by reason of the adhesions it is usually localized. In the present series four such cases proved fatal, no doubt because of the associated massive silicosis and cardiac embarrassment. The main bronchi show slight fusiform dilatation, but frank bronchiectasis is exceedingly rare. This recalls the fact that clinically clubbing of the fingers and toes is not a feature of silicosis.

In the early fine nodular stage of the disease, if the pleura is comparatively normal, a very characteristic post-mortem appearance is mammillation of the visceral pleura. Small sago pearls, tiny subpleural silicotic nodules with an areola of carbon pigment, protrude from the surface of the lung. The hilum glands are enlarged, fibrotic, rubbery, and deeply pigmented with carbon. As a broad generalization it may be said that if the root glands are not silicotic then one is unlikely to discover silicotic nodules in the lung parenchyma; on the other hand if the glands are involved it does not follow that characteristic lesions will necessarily be found in the lung tissue; if the lungs reveal nodules then the glands will also show fibrotic changes.

The mature silicotic nodule is usually described as gritty or shotty to the touch, but this is rather misleading in that it tends to suggest palpable aggregates of silica and does not convey the firm rubbery texture of the redundant fibrous tissue. Even so it has not the elasticity of the lung parenchyma, so that when a cut is made in the lung the nodules stand out on the cut surface and are visible as well as palpable.

In the massive infective type of silicosis the individual confluent nodules are not identifiable, and indeed one is impressed in such cases by the paucity of discrete nodules elsewhere in the lung. The centre of these masses is often excavated and the cavity is filled with black, oily debris closely resembling dirty motor lubricating oil. At times there is no actual defined cavity but the fibrotic tissue appears as if it had been gnawed by a mouse. There are many who consider these appearances indicative of tuberculosis. All such cases were carefully investigated, but in many the presence of tuberculosis could not be established even by

guinea-pig inoculation. The blood supply to the central area is often poor and, as revealed by histological examination, associated with vascular degeneration, so that ischæmic necrosis is a possible explanation. On the other hand it may be that the characteristic histological appearances of the tubercle are greatly altered in these cases and so are misinterpreted. While the classical silicotic nodule and the simple tubercle are well-defined entities familiar to the expert histologist, the combined lesion is not so readily identified and often leads to differences of opinion. This was confirmed when portions of the same material were submitted to a panel of expert pathologists.

TABLE 14

PRIMARY BRONCHIAL CARCINOMA : NO SILICOSIS
130 males : 20 females

No.	Sex	Age	Lung	Remote metastases	Occupation or process
1	F	40	L	None	General earthenware (clay)
2	M	51	R	None	General earthenware (clay)
3	M	47	R	None	Sanitary earthenware
4	M	59	R	None	Millman
5	M	60	R	None	Millman
6	M	43	R	Kidneys	Electrical porcelain
7	M	65	R	None	Electrical porcelain
8	M	47	R	None	China : flint
9	M	49	R	None	China : clay
10	M	56	R	None	General earthenware (clay)
11	M	53	R	Liver	General earthenware (clay)

Influenza and Pneumonia.—In certain cases of influenza the intense brick red coloration of the parietal pleura was very striking. Death from massive pneumonia, within a few days of onset, occurred from time to time, and necropsy followed within a few hours. In some of these cases the visceral pleura over the consolidation was raised in tiny blebs, of various sizes, like a coarse irregular herpetic eruption. The condition seemed to represent acute emphysema of superficial air vesicles.

Primary Bronchial Carcinoma.—During recent years a good deal of attention has been directed to the possible relationship of primary bronchial carcinoma and the inhalation of silica. Anderson and Dible (1938) submit that : "A group of cases of pulmonary cancer exists, in which the organs contain an excess of silica and show histological evidence of silicotic fibrosis. The conclusion we think is that the role of silica is ætiological."

In this series thirteen cases of bronchial carcinoma among 359 persons were noted in association with simple silicosis, while two cases occurred among the 241 patients who died of silicosis accompanied by tuberculosis. On the other hand eleven cases of bronchial carcinoma occurred among the 150 cases in which there was no evidence of silicosis, though it should not be overlooked that most of these workmen had long periods of exposure to the inhalation of silica, free and combined. On this evidence and the recorded experience of other observers, we are not sufficiently convinced to subscribe to the conclusion quoted.

Brief details in Tables 14, 15, and 16 may be of interest.

Dr. S. Roodhouse Gloyne undertook the histological examination and with the exception of a few cases of squamous or mixed cell carcinoma, all others were reported as oat-cell type.

Other Organs.—There was no convincing evidence of general systemic intoxication, with damage to organs or tissues apart from the lungs, which might be attributed to the inhalation and excretion of silica.

Silicotic fibrosis was occasionally observed in abdominal glands but, though search was made, we were never satisfied that silicotic nodules might occur in the spleen.

Summary

Silicosis is a serious and widespread disease among workmen engaged in the manufacture of pottery in the North Staffordshire area of Great Britain. Each year it causes the death of between forty and fifty persons. Between 1931 and 1946 the Stoke-on-Trent panel of the Medical Board for Silicosis attended the necropsy in 750 cases in which there was reason to suspect that death might have been caused by silicosis or silicosis accompanied by tuberculosis. The findings are analysed and tabulated. Among the subjects selected for special comment are : (1) cases occurring in china clay processes ; (2) tuberculosis in relation to silicosis ; (3) inanition ; (4) the heart and heart failure in silicosis ; (5) chronic nephritis ; (6) morbid anatomical changes in the lungs and pleura ; (7) primary bronchial carcinoma.

TABLE 15

PRIMARY BRONCHIAL CARCINOMA : SILICOSIS PRESENT
339 males : 20 females

No.	Sex	Age	Lung	Remote metastases	Degree of Silicosis	Occupation or process
1	M	56	R	None	Microscopic	Gen. earth : clay
2	M	70	R	Liver	Microscopic	Gen. earth : clay
3	M	64	R	Liver	Confluent	Gen. earth : clay
4	M	50	R	None	Microscopic	Gen. earth : clay
5	M	45	L	None	Miscrocopic	San. earth.
6	M	46	R	Suprarenals and parotid	Microscopic	Polisher
7	M	56	L	None	Confluent	Earth. tiles
8	M	71	L	None	Confluent	Millman
9	M	48	L	Liver	Microscopic	China : flint
10	M	64	L	None	Confluent	China : flint
11	M	64	L	None	Confluent	China : flint
12	M	57	R	None	Nodular	China : flint
13	M	60	R, L	Skull	Microscopic	Gen. earth. : clay

TABLE 16

PRIMARY BRONCHIAL CARCINOMA : SILICOSIS ACCOMPANIED BY TUBERCULOSIS
207 males : 34 females

No.	Sex	Age	Lung	Remote metastases	Degree of silicosis	Occupation or process
1	M	57	R	Liver	Confluent	Gen. earth.: clay.
2	M	58	L	None	Microscopic	Millman

This work has been made possible through the co-operation of my colleagues : Dr. Charles L. Sutherland, Chief Medical Officer of the Medical Board for Silicosis ; Dr. F. N. R. Price ; Dr. W. W. Jones ; Gerald W. Hunbach, Esq., H. M. Coroner ; H.M. Inspectors of Factories ; Dr. S. Roodhouse Gloyne ; the late Professor E. H. Kettle ; and my secretary, Miss Dorothea Hubank.

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SILICOSIS IN SANDSTONE WORKERS

SOME OBSERVATIONS BASED ON TWO HUNDRED AND SEVENTY-FIVE NECROPSIES

BY

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As a supplement to the preceding article on silicosis in pottery workers, a similar analysis has been applied to 275 necropsies occurring during the same period in several occupational groups, banker-masons, stone-dressers and cutters, metal-grinders, sandblasters, steel-fettlers, and coal-miners. All are exposed to the same hazard, namely, the inhalation of silica dust arising out of the use and manipulation of sandstone and its derivatives.

TABLE 1

DISTRIBUTION BY OCCUPATION

Occupation	No. of cases
Banker-mason	57
Stone-dresser and cutter	50
Metal-grinder	29
Sandblaster	26
Steel-fettler	7
Coal-miner	106
All occupations	275

Description of Occupations

Banker-mason.—This occupation comprises the shaping and dressing of rock, sandstone, limestone, marble, and granite for monumental purposes such as public buildings and bridges. The essential characteristic of the work is that each stone is fashioned by mallet and chisel to a size and design specified on an architect's blueprint. The masons in the group were employed in Cheshire, Staffordshire, and Derbyshire.

Stone-dresser and Cutter.—This work is similar to that described above but is restricted to dividing large blocks of stone and roughly shaping them by hammer and punch. The operations are frequently carried out at the quarries, and common products are wallstones, kerbs, setts, and grindstones. This group also is derived from the counties mentioned.

Metal-grinders.—This work involves the grinding of metals on grindstones composed of natural sandstone. The industry is mainly concentrated in Sheffield, but this particular group was employed in the manufacture of edge tools in and around Wolverhampton and Birmingham.

Sandblaster.—This group was employed at various foundries in the Midlands engaged in the cleaning of castings by means of dried quartzose sand.

Steel-fettler.—This work involves the freeing of steel castings from adherent sand by means of hand, pneumatic, or power-driven tools.

Coal-miner.—This group is restricted to underground workers, and so far as dust disease of the lungs is involved relates only to silicosis; dust reticulation is not considered. The men were employed in North and South Staffordshire.

TABLE 2

CERTIFIED CAUSE OF DEATH

Cause of death	No. of cases	%	Pottery survey %
Silicosis	76	27·6	28·9
Silicosis accompanied by tuberculosis	93	33·8	32·1
Other causes (silicosis present)	41	14·9	19·0
Other causes (no silicosis)	65	23·7	20·0
All causes	275	100·0	100·0

Analysis and Commentary

The 275 necropsies completed under the direction of H.M. Coroner were distributed as shown in Table 1. As in the previous survey the cause of death is related to silicosis or other causes (Table 2). The distribution in the two surveys is remarkably parallel (Table 3).

Silicosis Accompanied by Tuberculosis

In this context tuberculosis means active tuberculosis of the lungs with tubercle bacilli present in the sputum, and in ninety-three cases the combined disease, silicosis accompanied by tuberculosis, was the cause of death. The distribution of these by age group and occupation is set out in Table 4.

TABLE 3

CAUSE OF DEATH ACCORDING TO OCCUPATION

Occupation	Cause of death				All causes
	Silicosis	Silicosis + tuberculosis	Other causes (Silicosis present)	Other causes (no Silicosis)	
Banker-mason	17	21	12	7	57
Stone-dresser and cutter ..	15	18	12	5	50
Metal-grinder	12	11	4	2	29
Sandblaster	5	18	1	2	26
Steel-fettler	2	5	—	—	7
Coal-miner	25	20	12	49	106
All occupations	76	93	41	65	275

TABLE 4

AGE AT DEATH IN RELATION TO OCCUPATION

Age group	Occupation						All
	Banker-mason	Stone-dresser	Metal grinder	Sand-blaster	Steel-fettler	Coal-miner	
Under 20 ..	—	—	—	—	—	—	—
20–24	—	—	—	—	—	—	—
25–29	—	1	—	2	—	1	4
30–34	—	1	—	2	—	—	3
35–39	—	—	3	2	—	1	6
40–44	2	1	—	3	3	1	10
45–49	1	2	2	5	—	1	11
50–54	5	3	1	2	—	3	14
55–59	5	4	2	—	1	4	16
60–64	4	2	3	2	—	3	14
65–69	4	3	—	—	1	6	14
70 + ...	—	1	—	—	—	—	1
All ages ..	21	18	11	18	5	20	93

While the high incidence of tuberculosis in late middle life again appears (Table 5), the occurrence of cases in sandblasters under the age of 40 years is serious, a matter which has been remarked upon by Merewether (1936) and which appears in th Annual Reports of H.M. Chief Inpsector Factories (1933 *et seq.*).

TABLE 5

DEGREE OF SILICOSIS ASSOCIATED WITH FATAL TUBERCULOSIS

Occupation	Degree of silicosis			Total
	Micro-scopic	Nodu-lar	Con-fluent	
Banker-mason ..	3	7	11	21
Stone-dresser ..	1	7	10	18
Metal-grinder ..	2	5	4	11
Sandblaster ..	4	8	6	18
Steel-fettler ..	—	2	3	5
Coal-miner ..	1	6	13	20
All occupations	11	35	47	93

It is confirmed that fatal tuberculosis may emerge even when the degree of silicosis is minimal though most commonly the association is with massive fibrosis. Sandblasters again appear exceptional, but the absolute numbers are too small to justify any definite conclusions. As shown in Table 3, there was evidence of silicosis in 210 cases, and in ninety-three cases this was combined with active tuberculosis, which represents an incidence of 44·3 per cent.; the corresponding figure among pottery workers was 40 per cent. This contrasts with the recorded opinion of authorities in Great Britain (Bridge, 1931; Sutherland, 1945; Hale, 1945), South Africa (Simson and others, 1930), and France (Deffrenne, 1945), who place the incidence between 46 and 75 per cent.

In view of the tentative explanation advanced in respect of pottery workers it appeared worth while to relate tuberculosis in silicosis with age incidence, but to ensure uniformity it was deemed advisable to restrict the analysis to male workers. The analysis covers the pottery workers (P) and the miscellaneous group of occupations (M) (Table 6).

The diminishing influence of tuberculosis as the cause of death with the advance of age is apparent and supports a hypothesis which was formulated

TABLE 6

FATAL TUBERCULOSIS COMPLICATING SILICOSIS

Age group	Silicosis			Other causes (Silicosis present)			Silicosis + tuberculosis			All cases with evidence of silicosis	% Silicosis + tuberculosis to silicosis
	P	M	Total	P	M	Total	P	M	Total		
Under 20 ..	—	—	—	—	—	—	—	—	—	—	—
20–24	—	—	—	—	—	—	—	—	—	—	—
25–29	—	—	—	—	—	—	—	4	4	4	100·0
30–34	—	1	1	—	1	1	2	3	5	7	71·4
35–39	—	3	3	—	1	1	5	6	11	15	73·3
40–44	7	7	14	5	1	6	22	10	32	52	61·5
45–49	7	4	11	10	3	13	16	11	27	51	52·9
50–54	27	21	48	17	11	28	43	14	57	133	43·1
55–59	39	17	56	22	8	30	33	16	49	135	36·3
60–64	52	10	62	37	10	47	36	14	50	159	32·1
65–69	51	8	59	23	2	25	36	14	50	134	37·3
70+	18	5	23	24	5	29	14	1	15	67	22·4
All ages ..	201	76	277	138	42	180	207	93	300	757	39·7

TABLE 7

PRIMARY BRONCHIAL CARCINOMA
(NO SILICOSIS)

No.	Initials	Occupation	Age	Lung	Remote metastases	Remarks
1	W. N.	Metal grinder	47	R	Liver	T.B. plus
2	E. S. G.	Banker-mason	67	R	Liver	—
3	J. G. G.	Collier	54	R	None	—
4	G. F. B.	Collier	57	L	None	—
5	J. H. S.	Collier	62	R	Liver	—
6	E. W. G.	Collier	39	R	None	—

TABLE 8

PRIMARY BRONCHIAL CARCINOMA
(SILICOSIS PRESENT)

No.	Initials	Occupation	Age	Lung	Remote metastases	Remarks
1	A. T.	Banker-mason	53	R	None	—
2	A. M.	Banker-mason	56	R	None	—
3	J. M.	Banker-mason	56	L	None	—
4	H. E. M.	Metal grinder	52	R	None	T.B. plus

from clinical observation. At the same time it helps to reconcile the divergent opinion of authorities. Maybe the real value is that the details may provide comparative data for other workers concerned with the cognate problems of the relationship of tuberculosis to asbestosis and dust reticulation of coal-miners, problems which are of vital practical importance and not merely of academic interest.

As to the further analyses of this series of 275 necropsies, precisely similar methods have been applied as were used for the 750 necropsies in pottery workers. The results coincide so closely that no useful purpose would be served by recording them in detail.

Primary bronchial carcinoma, however, is a subject of considerable interest to many observers at the present time so the details may be justified for reference.

In sixty-five cases death was due to "other causes" without any evidence of silicosis, and among these were six cases of primary bronchial carcinoma; in one it was associated with active pulmonary tuberculosis (T.B. plus) (Table 7).

Among the 210 cases in which there was evidence of silicosis in some degree were four cases of primary bronchial carcinoma, of which one was associated with silicosis accompanied by tuberculosis (T.B. plus) (Table 8).

Summary

In a previous communication an analysis was made of 750 necropsies in pottery workers with special reference to silicosis and related problems.

This is supplemented by a similar analysis of 275 necropsies in a miscellaneous group of occupations involving exposure to siliceous dust produced in the manipulation and use of sandstone and its derivatives.

Further attention is directed to tuberculosis occurring as a complication of silicosis, and details are given of ten cases of primary bronchial carcinoma.

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INHALATION RISK AND PARTICLE SIZE IN DUST AND MIST*

BY

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Respiratory Absorption and Elimination

The human respiratory system has long been known to filter the air passing through it. Lord Lister demonstrated that putrefactive organisms were not present in air passing out of the lungs and in 1869 Tyndall (1881) found that air expelled towards the end of a breath failed to produce scattered light near the focus of a light beam and caused the white track of the beam, due to atmospheric dust, to disappear. These early observations are incomplete: as a rule, when dusty air is inhaled, only a proportion of the dust is retained in the body while the rest is expelled. The particles which are left behind deposit in different regions of the breathing tract according to their size. The coarsest get trapped in the nose in normal breathing,

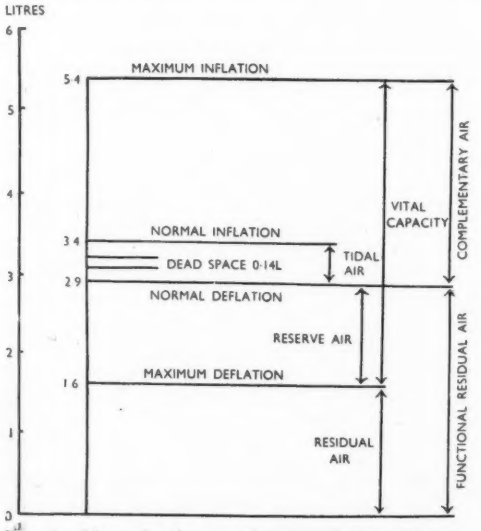

FIG. 1.—Normal volume and nomenclature of human lungs.

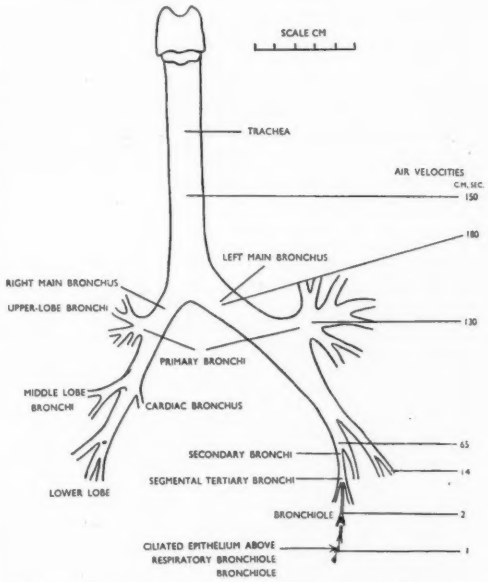

FIG. 2.—Scale diagram of respiratory channels of human lung.

which is always through the nose in contaminated atmospheres, save during heavy exercise. Very fine particles penetrate deeply into the lungs and a considerable number are exhaled.

Fig. 1 is a diagram (based on measurements of Aslett and others, 1939) which shows to scale the volumes of air associated with respiration and explains the usual nomenclature. It can be studied in conjunction with fig. 2, a scale diagram of the human lungs, and the Table, which gives dimensions. Fig. 3 and 4 illustrate the nasal passages.

* A shorter version of this paper was presented at the Second International Congress of Crop Protection, London, July, 1949.

The trachea, bronchi, and bronchioles are lined with a continuous ciliated epithelium (fig. 5) which terminates at the entrance to the respiratory bronchioles. The cilia engage with a film of mucus which they keep in steady upwards movement at a rate rising to 3 or 4 cm. per minute in the trachea. Peristaltic movements and coughing may co-operate to provide a mechanism which has been shown by Barclay and others (1942) to be very competent in lifting dust particles up to the pharynx, when they can be swallowed, sneezed out, or expectorated. It is less effective for liquids, which may slip down into the alveoli, though fine mist droplets should be eliminated satisfactorily if the concentration is low. The ciliary movement is ineffective if the mucus is not of the right consistency owing to illness or pharmacological action.

Particles of solid which reach the alveoli are passed into the lymphatic system or ingested by mobile phagocytes which may transport them to the mucus escalator. This process has been reviewed by Robertson (1941).

TABLE

DIMENSIONS OF THE HUMAN LUNG

Part of Lung	Number	Length	Internal Diam.	Volume
Trachea	1	12 cm.	1·4–2·0 cm.	14·3 cm.³
Main bronchi ..	2	2·5–5	1·0–1·6	7·1
Primary bronchi ..	12	3	0·4	4·5
Secondary bronchi	100	1·5	0·2	4·6
Tertiary bronchi ..	770	0·5	0·15	7·0
Bronchioles ..	54,000	0·3	0·1–0·2	4·5
Respiratory bronchioles ..	110,000	0·15	0·05–0·1	33
Alveolar ducts ..	26,000,000	0·02	0·02	164
Alveolar sacs ..	52,000,000	0·03	0·03	4,320

A further protective factor, purely physical, was suggested by Aitken (1883–4). Air entering the breathing channels is cooler than the body and relatively dry. A force of thermal repulsion therefore exists to keep particles away from the walls of the channels, and this is supplemented by a continual stream of water molecules, evaporating from the surfaces, which bombard dust particles and tend to drive them back. Thus the retention of fine particles in the lungs would be enhanced by high temperature and humidity.

Site of Action

The respiratory tract is protected with remarkable efficiency against inert, dry solids deposited anywhere save in the very depths of the lung. Elimination is less effective from these alveolar regions, of considerable surface area (some 15m²), which constitute, anatomically, the respiratory unit where oxygen absorption takes place. Liquids, or suspensions of powder in liquid, may reach the alveoli irrespective of the location of their first contact with the walls of the breathing system. This fact, associated with impairment of the ciliary motion, may explain the occasional deaths reported from exposure to high concentrations of mists which are not highly toxic; especially is this common in the aged. High concentrations of relatively inert dust might equally be dangerous to individuals suffering from loss of function of the ciliated epithelium. Irritant dusts which are too coarsely particulate to reach the alveoli in a healthy subject might, nevertheless, penetrate so far if chemical interference with the mucous blanket should occur.

At the same time it must be remembered that serious symptoms can arise without implication of the alveoli. Water-soluble substances and gases, for example, are rapidly absorbed in the bronchi. Again, small animals can be killed with some gases which operate as systemic poisons. The lungs are unharmed but the nasal passages become inflamed, indicating the site of absorption. Other gases destroy the lungs. The difference depends on solution in the nose. If rapid, and unimpeded by accumulation of reaction products, the poison may be entirely absorbed from the inhaled air while traversing the nose. This is less likely to occur in large animals or man and shows the need for care in estimating dangers to human beings from experiments with small animals. Nevertheless, large liquid or solid particles trapped in the nose could cause local or systemic injury in man without lung damage. Thus, coarsely particulate clouds of arsenicals of the blue cross type, as used by the Germans in the world war 1914-18, produce unpleasant symptoms in the nose. A particle size of about 30 μ (microns) could be obtained by dispersing with explosive or by spraying. On the other hand fine clouds of particles below 1 μ, generated by thermal evaporation, cause acute chest pains and, incidentally, penetrate poor-quality gas masks quite readily.

Men and animals after a lifetime of exposure to wind-blown desert sand have been found to possess lungs unscarred by silicotic lesions, while a few years' exposure to particles of the same chemical constitution, which have been released from sandstone grinding-wheels or in the figuring of millstones, may be disastrous. The difference is due to particle size. Desert sands are very much coarser than the dust released by the vigorous action of tools upon sandstone.

We see, therefore, that particle size is decisive in governing the region of initial deposition in the respiratory tract but that only in certain cases can immunity from hazard be claimed on this basis.

FIG. 3.—Section of human nose and pharynx.

FIG. 4.—Section of human nose.

However, an understanding of the role of particle size in a purely mechanical way is of some value in discussing inhalation risks.

Experiments on Inhalation and Retention of Dusts

Following the observations of Lister and Tyndall the dust content of exhaled air was examined more carefully by Owens (1923), using his new dust-counting apparatus. He showed how the retention of dust depended on the method of breathing. A subject breathing dusty air by normal respiration, using his tidal air, had very little dust in the reserve air (fig. 1) when it was sampled after a deep exhalation. On the other hand, if the reserve air was sampled after deep breathing for several cycles, its dust was only reduced to three quarters of the original content of the atmosphere.

The alveolar air, in the first case, was pure because the exchange between tidal air and alveolar air is mainly by diffusion with only slight mechanical mixing, so that particles do not readily gain access to the alveoli. In the second case, deep air is forced in and out and particles are taken right down where some can deposit by sedimentation and Brownian motion.

Early experiments on particle retention were reviewed by Drinker and others (1928), and they themselves found, with widely differing dusts, that about 55 per cent. was held in the respiratory organs. The percentage was independent of concentration. Brown (1931) continued the work and claimed a tendency towards greater retention at low concentrations, which is rather difficult to account for. Breathing in through the mouth instead of the nose caused a decrease of about 10 per cent., which illustrates the greater filtration efficiency of the nose in comparison with the mouth. He also showed during exercise, or when 5 per cent. carbon dioxide was inhaled along with the dust to increase the depth and rate of respiration, that a greater proportion of inhaled dust escaped on breathing out. A similar result is found for some gases; with quick breathing and enhanced minute volume the time allowed for settlement or diffusion to the lung surface is diminished. Owens' observation suggests, however, that such dust as deposits during rapid, deep breathing may be located where it can do far more damage than higher up, where the prospects of elimination are better. The effect of particle size was not examined in detail, though Brown observed that airborne paticles were often aggregates containing numbers of the ultimate grains of dust, and that they were therefore more readily trapped in the upper respiratory passages than individual grains.

Previous workers had assumed their dusts to be completely dispersed, but the large quantities caught in the nasal channels suggest that this was not the case. This criticism applies to the work of Lehmann (1935), who measured the filtration efficiency of the nose and found generally high values, exceeding 80 per cent. in some of his subjects, although the particle sizes quoted in his paper range from 0·2 to 5 μ. Probably his method of sampling did not reveal large aggregates. Lehmann claimed that men suffering from silicosis had less efficient nasal filtration than healthy individuals. Since silicosis is due to very fine dust which penetrates the nose freely, this finding is difficult to interpret.

Tourangeau and Drinker (1937) also measured nasal filtration of limestone dust, using unidirectional flow, and obtained figures below 30 per cent. They demonstrated that nasal filtration efficiency was related to the resistance to air flow through the nose. The particle size is not given but many particles about 10 μ in size must have been present.

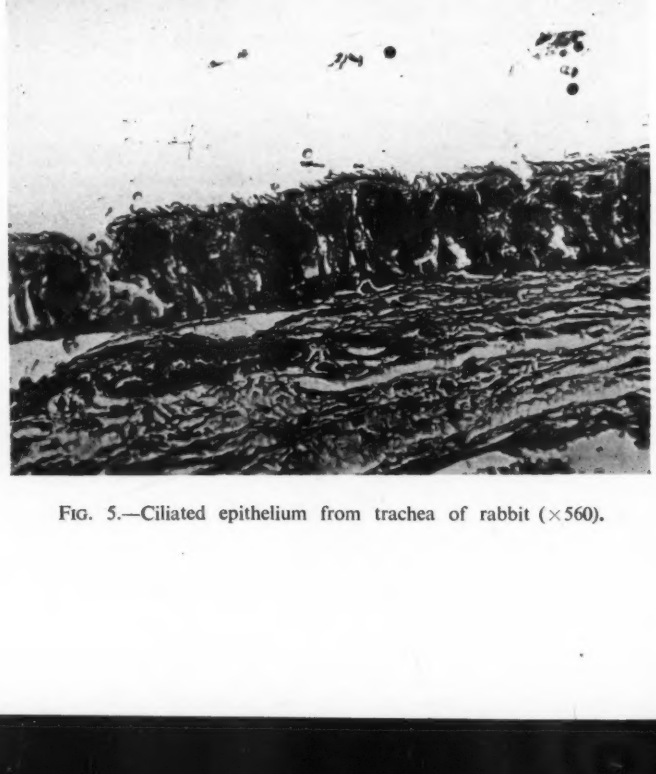

FIG. 5.—Ciliated epithelium from trachea of rabbit (×560).

The Effect of Particle Size

A theoretical estimate of the removal of airborne particles in the lung was made by Findeisen (1935) and is of great interest. He estimated the dimensions of various parts of the lung and, assuming a constant air flow of 200 c.cm./sec. during inhalation and exhalation, calculated the average air velocity at different points in the breathing tubes from the cross section and volume. His velocities are shown in fig. 2. This made it possible to judge the time which a given element of air spent in traversing the various air passages. The precipitation of particles on to the lung surfaces was considered to take place by four distinct mechanisms, namely: inertia, sedimentation, wall effect, and Brownian motion.

Inertia deposition, or impingement, occurs wherever the air passages change direction; owing to their mass, particles tend to continue moving in a straight line and may therefore come into collision with the bounding surface of the air channel. It is more effective for large, heavy particles and occurs particularly at the angles between bifurcations of the trachea and bronchi. The sensory receptors from which the cough reflex arises are concentrated at these points. Findeisen considered that this led to deposition, principally, for particles greater than 5 μ radius at the forks of the primary, secondary, and tertiary bronchi. Impingement is favoured by high air velocity.

Sedimentation operates where air speeds are low, in the deeper parts of the lung; no particles greater than 5 μ radius could reach the alveoli because they are removed by impingement or settlement higher up.

Wall effect is due to the particle being comparable in size with the air passage and reduces the effective size of the latter. It is important for elongated particles.

Brownian motion causes particles to diffuse towards a surface on which they precipitate. It is an appreciable factor in alveolar retention for particles below 0·3 μ radius and is the cause of the absorption of extremely fine particles in the bronchioles. Elsewhere, diffusion has no significance except for gases and in connexion with the humidification of inspired air.

Since experimental data on lung retention as a function of particle size is now available it is possible to check Findeisen's results and confirm the general accuracy of his views. Brownian motion is dependent only on the size of the particles and must lead to almost complete retention of sufficiently fine clouds. Sedimentation and impingement both increase with the quantity ρr^2 where ρ is the effective density of the particle and r is the radius. Since the particles may be open in structure, the effective density is often less than that of compact material. Hygroscopic solutions which absorb water may have both ρ, and especially r, increased during their passage into the lung and so have a greater chance of being deposited and a smaller chance of reaching the alveoli. Apart from this, the deposition of particles of different materials will be the same for those sizes over which settlement and impingement are the chief mechanisms if the quantity ρr^2 is held constant.

Experiments on the retention of particles in the lungs have been carried out by Van Wijk and Patterson (1940), by Landahl and Herrmann (1948) and by Wilson and La Mer (1948). The methods used by these workers were quite different.

The first named dispersed a cloud of quartz dust and sampled the original and exhaled clouds by means of thermal precipitators. From comparative size distributions, determined by counting under the microscope, the percentage removed by breathing was calculated as a function of particle size. Since quartz has a density equal to 2·5 these results have been reduced to unit density by multiplying the radius by $\sqrt{2{\cdot}5}$ so that comparison with the other results and with Findeisen's calculations is facilitated. This is justifiable over most of the range of sizes which they cover where impingement and sedimentation are the principal mechanisms at work. For the smallest sizes it may lead to a slight overestimate of particle retention which is of no significance.

The experiments of Landahl and Herrmann demonstrate that particles of high density are retained to a greater extent and that hygroscopic particles absorb moisture to form drops, which are larger and are more effectively retained than the original particles would have been. Their results for droplets of corn oil of density 0·91 are considered below.

Wilson and La Mer worked with particles of nearly uniform size formed from glycerol by vaporization in a special apparatus. Each particle contained a trace of radioactive sodium Na^{24}. By adjusting the generator, the drop size could be varied from 0·2 μ to 2·6 μ radius. A number of subjects inhaled the aerosols and measurements were made from which it was possible to deduce the total quantity retained in the respiratory tract and also the amount in the alveoli.

The observations consisted of β and γ ray activity determinations with a Geiger counter. The total retention was estimated from the deposit in filters through which measured volumes of inhaled and exhaled air had been drawn. To find the alveolar retention the counter was screened with a lead shield containing a slot. It was placed

against the chest wall, in the axillary region, with the slot horizontal and cutting across the fifth rib. The β radiation was absorbed by the body but the γ rays penetrated and could be counted. Since the counter was shielded from the upper respiratory tract and was remote from the larger air spaces, the radiation picked up came predominantly from the peripheral portion of the lung near the chest wall. These observations gave information about the relative retention in the alveoli as a function of particle size; to gauge the absolute retention additional measurements were made of count frequency from a standard source and also of counts from blood samples.

The experimental results for total percentage retention, P, are expressed by the following equation:

$$P = 105r^{0.163} - (2.37 - 0.29r)\,R$$

where r is the particle radius in microns and R is the frequency of respiration, in cycles per minute, ranging from 5½ to 20. The formula must not be used outside the ranges of the variables covered in the experiments. The term in brackets is given incorrectly in the original paper.

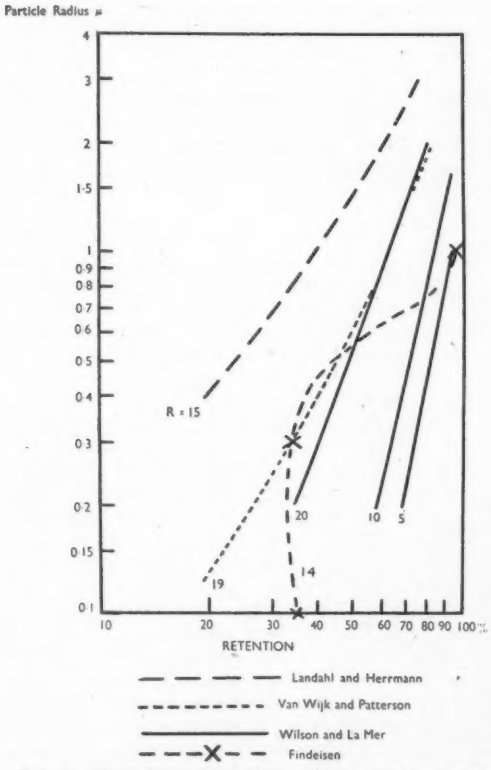

FIG. 6.—Retention of particles of unit density in the lungs. R = breathing cycles/min.

The retention is greater for slow breathing, in agreement with the observations of previous experimenters. It was also noticed that deep inspiration, followed by a brief pause before exhalation, caused a greater retention of small particles. The alveolar retention exhibited two maxima with peaks in the range 0·3 to 1·0 μ radius. The maximum alveolar retention was about 45 per cent. of the inhaled aerosol.

In fig. 6 the theoretical results of Findeisen are exhibited in comparison with the available experimental data. Those of Van Wijk and Patterson and of Landahl and Herrmann have been reduced to particles of unit density as explained above. The experiments of the latter workers included various kinds of particulate material and the results for corn oil were selected for this comparison because the particles are non-hygroscopic, spherical droplets and should be influenced only by mechanical factors. Their subjects breathed 15 cycles per minute using 450 c.cm. tidal air. The lung retention is substantially less than in the other work, and it is suggested that an over-estimate of particle size, which can easily occur with oil droplets, might account for the discrepancy. With other materials higher retentions were measured.

The curve from the results of Van Wijk and Patterson, at 19 cycles per minute, is very close to that of Wilson and La Mer for 20 cycles, while a smooth curve through Findeisen's theoretical points (14 cycles per minute) is also in quite good agreement. Findeisen appears to predict too high a retention for both smaller and larger sizes. According to his theory the retention of particles of 1 μ radius takes place to the extent of 82 per cent. in the alveoli, and 15 per cent. in the bronchioles. However, as we have seen, the exchange between tidal air and alveolar air is quite small in normal respiration, a fact which Findeisen did not take sufficiently into account in his treatment. As a result he reckoned on too great a penetration of 1 μ particles to the alveoli and so overestimated their retention. For the same reason his estimate for the retention of particles about 0·1 μ radius is probably high.

Fig. 7 exhibits a comparison between the average alveolar retention, from the experiments of Wilson and La Mer, and the theoretical alveolar retention according to Findeisen. The experimental data shows the double peak already referred to; in the curves for the seven individual subjects, which are here shown averaged, the peaks were, of course,

more pronounced. Wilson and La Mer consider that their curve includes some deposition in the finer bronchi and bronchioles. Findeisen's computed figures for the alveoli and also for the alveoli plus bronchioles are shown. It seems that Findeisen overestimates retention in the finer air passages for particles of about 1 to 2 μ radius and underestimates retention for radii of the order of 0·4 μ which takes place, in his view, almost entirely in the alveoli. It is probable, therefore, that sedimentation in the fine bronchioles is rather more important than he considered for particles of this size.

On the whole the general picture drawn by Findeisen is well substantiated so it is evident that

Nasal Filtration

Deposition in the nose is mainly due to impingement, owing to the inertia of the particles. This is shown by the increase in retention which follows an increase in the rate of air flow, and has been demonstrated in small animals and man by Davies (1946) and by Boyland and others (1947).

Landahl and Black (1947), and later Landahl and Tracewell (1949), studied the penetration of airborne clouds through the human nose by drawing the cloud up the nose and out through the mouth while the subject held his breath. The particle concentrations in the original and passing clouds were compared by analytical methods. Reduction

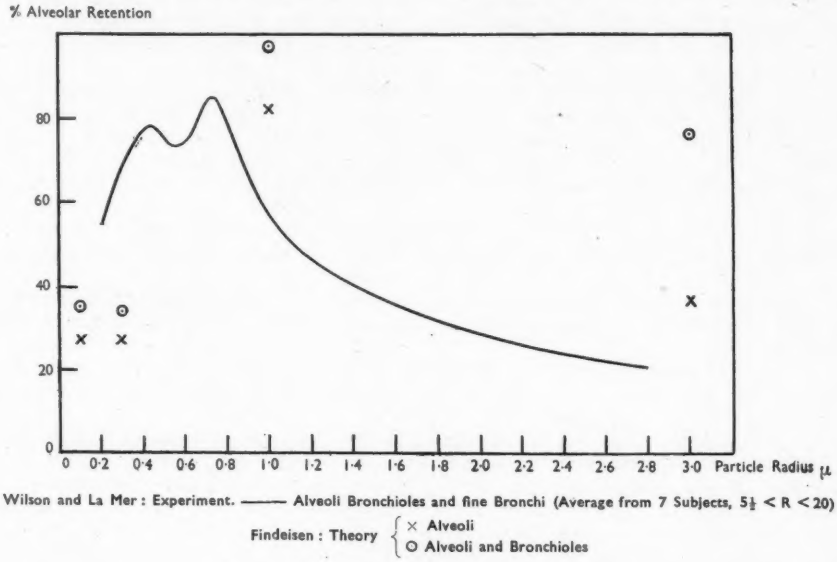
% Alveolar Retention
80
60
40
20
0
0 0·2 0·4 0·6 0·8 1·0 1·2 1·4 1·6 1·8 2·0 2·2 2·4 2·6 2·8 3·0 Particle Radius μ
Wilson and La Mer: Experiment. —— Alveoli Bronchioles and fine Bronchi (Average from 7 Subjects, 5½ < R < 20)
Findeisen: Theory
× Alveoli
O Alveoli and Bronchioles

FIG. 7.—Retention of particles in the deep lung.

thermal and evaporative effects, considered by Aitken (1883-4), can be of no great significance in decreasing lung retention.

Landahl and Herrmann show that the minimum retention in the lung takes place for particles about 0·1 to 0·15 μ radius. Retention increases for still smaller particles owing to Brownian motion. Although the minimal retention is quite low the significance of particles of this size may be considerable since they have access to the alveoli. Thus Hatch and Kindsvatter (1947), using fine quartz dust with a median radius of 0·09 μ, determined with the electron microscope, produced lung changes in guinea-pigs after two weeks' exposure to a concentration of 35 mg./m³.

of their findings for different materials to spherical particles of unit density shows that 80 per cent. penetration occurs for particles of radius 0·9 μ, 50 per cent. at 2 μ, and 20 per cent. at 6 μ. Compact spherical particles of high density will be caught more readily in the nose, while loose, flocculant aggregates will penetrate to a greater extent than is suggested by the figures quoted. These must, of course, be subject to considerable variation according both to the individual and to the circumstances. The turbinate bones in the nose are covered with erectile tissue which may become swollen with blood so that they partially block the airways. Cold, dry air promotes this reaction while warm, moist air causes shrinkage. The complexity of

the nasal channels in some small animals, together with the low rate of air flow, leads to the air becoming saturated with moisture during its passage through the nose; this is not the case in man.

Discussion

We have seen that particles down to about 6 μ radius are retained in the nose. With mouth breathing they would hardly penetrate deeper than the secondary bronchi. Deposition of these and larger particles is mainly due to impingement. At 2 μ radius penetration of the nose is considerable and most particles are deposited by sedimentation in the bronchioles; a small proportion reach the alveoli. Above 2 μ scarcely any particles are exhaled. Maximal deposition in the alveoli and fine bronchioles occurs between 0·4 and 0·8 μ radius and is also due to sedimentation. Some 80 per cent. of particles of radius 0·1 to 0·15 μ are breathed out again, this being the range over which retention is a minimum. Deposition in this range takes place almost entirely in the alveoli. Still smaller particles deposit, on account of their Brownian motion, in the alveoli and bronchioles.

Retention is enhanced by slow breathing because it allows more time for sedimentation of particles on to the surfaces of the bronchioles and alveoli. Alveolar retention is enhanced by deep breathing because exchange between tidal and alveolar air is then more effective.

The figures quoted relate to spherical particles of unit density. For other densities the radii given above should be divided by the square root of the density of the particle which may be several times smaller than the density of the material dispersed. Thus particles of low density penetrate further. This does not apply for very small radii, below 0·1 μ, where Brownian motion is important, since this is independent of density. Particles which deviate from spherical shape will penetrate to greater depths, since the surface to mass ratio is greater; clearly, however, this may not be the case for extreme shapes, such as fine fibres, which are readily entangled.

Apart from retention, the solubility of particles is an important factor influencing the risk from dust exposure. Having quartz dust in mind, Hatch and Hemeon (1948) showed the existence of a most dangerous size on the assumption that solubility increases as particle size diminishes. This is in accord with experiment; it is difficult to demonstrate that a pebble of quartz dissolves at all, but finely powdered $Si\,O_2$ readily forms a 0·01 per cent. to 0·015 per cent. solution. On combination of this effect with the lung retention factor it was shown that particles of about 0·5 μ radius are the most toxic. A dust with a median number diameter of 0·5 μ was twelve times more active than a dust with a 1·5 μ median. This calculation was based on alveolar retention. Substances of greater solubility than quartz might be dangerous when deposited in the higher regions of the respiratory tract.

Summary

Particles greater than about 6 μ (microns) radius are retained in the nose. With mouth breathing they would not be deposited below the secondary bronchi. Deposition of these and larger particles is mainly due to impingement. At 2 μ radius penetration of the nose is considerable and most particles are deposited by sedimentation in the bronchioles; a small proportion reach the alveoli. Above 2 μ scarcely any particles are exhaled. Maximal deposition in the alveoli and fine bronchioles occurs between 0·4 and 0·8 μ radius and is also due to sedimentation. Some 80 per cent. of particles of radius 0·1 to 0·15 μ are breathed out again, this being the range over which retention is a minimum. Deposition in this range takes place entirely in the alveoli. Still smaller particles deposit, on account of their Brownian motion, in the alveoli and bronchioles.

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Particles deposited in the trachea, bronchi, or bronchioles are normally removed by ciliary action; this will not prevent absorption of water-soluble material and may fail through impairment of the ciliated epithelium or the mucus coating due to illness or chemical action. The ciliary mechanism is ineffective for liquids. Elimination from the alveoli is by phagocytes. Although particle size is decisive in governing the region of initial deposi-

tion in the respiratory tract, only in certain cases is it possible to claim immunity from health risk on this basis.

The author has made free use of a private communication from Professor J. H. Gaddum, F.R.S., on the retention of inhaled clouds ; he is indebted to W. A. Fell, Esq., and R. H. D. Short, Esq., for the loan of anatomical specimens, and to Dr. T. Bedford for valuable advice and encouragement.

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PHOTOELECTRIC MEASUREMENT OF COAL DUST STAINS ON FILTER PAPER

BY

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Introduction

Owing to the rapidity with which observations can be made, assessment of airborne dust by aspiration through filter paper, followed by photoelectric measurement of the resulting stain, has been very popular. The following account of experiments with coal dust shows how the calibration depends on particle size, and the low sensitivity of the method for small particles, when large ones are also present, is exposed. The work was carried out with the hand-pump and densitometer developed by the Pneumoconiosis Research Unit of the Medical Research Council, which has been described by Watson (1948) and by Watson and Hounam (1948). The increase in resistance to air flow through the filter paper, due to deposition of coal dust, was also studied.

Experimental Method

The coal used was dry duff, the fines from screening anthracite, which yielded 18 per cent. ash and is representative of general air dust. It was ball-milled dry, using one-inch pebbles for twenty hours, and sieved down to 200 mesh.

A known weight of dust was passed into a chamber of 189 litres, through an elutriator, under carefully controlled conditions which kept the aggregation of particles down to a minimum. During dispersal and sampling the dust cloud was fanned with a thin sheet metal propeller of low pitch, nine inches in diameter and rotating once a second.

The particle size was controlled by varying the flow through the elutriator during dispersal and by varying the interval between the termination of dispersal and the commencement of sampling from one minute to sixty minutes. During this time the cloud was not fanned.

The density of the airborne particles ranged from about 1·4 g/cm.^{3} in the case of the smaller units up to about 0·4 for the larger aggregates (Davies, 1947).

Sampling was carried out in comparison with absolute samples taken with the sedimentation cells described elsewhere (Davies, 1946). In the case of the finest cloud these had been shown to collect, over all sizes 15 per cent. more particles than the thermal precipitator.

The diameter is that of a circle equal to the projected area of a particle as seen under the microscope.

The apparatus was arranged in the chamber before dispersing the dust. At the appropriate time a sedimentation sample was taken; then the hand pump was operated for the requisite number of strokes; and, finally, a second sedimentation sample was withdrawn. The average of the sedimentation counts was used to establish the dust concentration and size distribution.

A series of experiments was performed within which the cloud was reproduced as exactly as possible. The number of strokes of the hand pump differed, however, so that the amount of dust deposited on the filter paper was variable. The number of dust particles per cm.^{3} being N and the number of pump strokes n, the product Nn measures the quantity of dust which should be caught on the filter paper. Watson has shown that a linear relationship exists between Nn and $100D^{1\cdot5}$, where D is the optical density of the coal dust deposited on the paper. The galvanometer in the P.R.U. densitometer, in addition to reading microamperes, is scaled to read $100D^{1\cdot5}$, starting at zero for full scale deflection (50 microamps.) and reading infinity for zero current. The maximum reading which can accurately be made is 100. The scale is graduated on the assumption that the deflection is proportional to light intensity. Hence, if i is the current in microamperes, I the intensity of transmitted light incident upon the photocell, and I_0 the original intensity, we have $D = \log_{10}(I_0/I)$ and $I_0/I = 50/i$.

Putting $R = 100D^{1\cdot5}$ = galvanometer reading when R = 0 for clean filter paper, it was confirmed that

$$Nn = c\,R \qquad (1)$$

for a given cloud, but c is a function of particle size which increases for finer particles.

It was found, in addition, that a linear relationship existed for a given cloud between Nn and P, the increment in pressure drop, due to coal dust, when measured across the filter paper with an air flow of one litre per minute. Hence,

$$Nn = c'\,P \qquad (2)$$

The pressure is proportional to the air flow over a considerable range within which the above figure falls.

A preliminary determination of the variability of clean filter paper (Whatman No. 1) was made. The densitometer was set to give a low reading with clean paper, so that all samples would give values of R falling on the scale. Forty pieces of paper gave a mean value $R = 1{\cdot}09$ and a standard error of 0·31. The same pieces had a mean pressure drop P at one litre/min. of 3·51 cm. mercury and a standard error equal to 0·17.

It was clear from these findings that the variation of the filter paper was so great that it was desirable to set the densitometer to read zero with the actual paper used in an experiment, except when the deposit was dense, and also to determine the pressure drop across each clean paper individually.

In a given series of experiments, that is to say with nearly constant cloud, the values of c and c′ in equations (1) and (2) were determined by the method of least squares supposing that no error existed in Nn. From the different series, and by combining the results as explained below, the dependence of c and c′ on particle size was made evident.

The Coal Dust Clouds

Four series of experiments, A to D, were done with clouds of increasing fineness. The same weight of coal dust was dispersed in every experiment, but the finer clouds had lost more particles by settlement, either inside the elutriator or during the interval between dispersal and sampling. We can consider cloud A as basic and regard the others as modifications due to the removal of increasing proportions of the coarse end of the size distribution.

Let us take, for example, the coarsest cloud, A, and the finer cloud, B. The letters will serve as suffixes to identify measurements relating to each cloud. We want to deduce the value of the constant c_{A-B} (equation 1) for the theoretical difference cloud which contains only the coarse particles of A. It consists of the particles which settled from A leaving B behind. c_A and c_B are known from the experimental work on A and B.

Following a discussion by Walton (1947), work by Vouk (1948) in this laboratory has shown that light screening by coal dust particles greater than one micron is nearly proportional to their total projected area of cross section, that is to the product of the mean of the squares of the diameters with Nn, provided that the angle of acceptance of the photocell which measures light intensity is great enough to collect all the light diffracted by the particles. This is approximately the case in the P.R.U. densitometer.

However, light absorption by particles on filter paper is proportional to their total area only when the number in the deposit is small. At higher optical densities less light is cut off than would be expected on this basis because the particles overlap and the screening area is diminished. Armitage (1949) has shown that the frequency of occurrence of clumps can be expressed quite well as a function of the number of particles per unit area and the mean of the squares of their diameters, $\overline{d^2}$. Correction terms which involve higher moments of the distribution of sizes are small, and the result is probably not very sensitive to particle shape. Also, the mean area of overlap in all possible arrangements of particles in a clump is a function of $\overline{d^2}$.

Hence, it is evident that the light-screening effect, R, must be expressible in terms of Nn and $\overline{d^2}$, providing the particle shape does not depend too much upon size.

The constant c of equation (1) must therefore be a unique function of the mean of the squares of the diameters. We can therefore write :

$$R_A = (Nn)_A \, f\,(\overline{d_A^2}) = (Nn)_A \,(\alpha + \beta\, \overline{d_A^2} + \ldots)$$

$$R_B = (Nn)_B \, f\,(\overline{d_B^2}) = (Nn)_B \,(\alpha + \beta\, \overline{d_B^2} + \ldots)$$

If the same number of pump strokes were made in sampling clouds A and B, then, for the theoretical difference cloud,

$$(Nn)_{A-B} = (Nn)_A - (Nn)_B$$

so that

$$R_{A-B} = \{(Nn)_A - (Nn)_B\}\, f \left\{ \frac{(Nn)_A\, \overline{d_A^2} - (Nn)_B\, \overline{d_B^2}}{(Nn)_A - (Nn)_B} \right\}$$

If the function f is taken as linear over the limited range between $\overline{d^2}_{A-B}$ and $\overline{d^2}_B$ it is easy to see by expanding the last expression that

$$R_{A-B} = R_A - R_B$$

Also, from equation (1)

$$c_{A-B}\, R_{A-B} = c_A\, R_A - c_B\, R_B$$

Eliminating R_{A-B} between the last two equations gives

$$c_{A-B} = \frac{c_A\, R_A - c_B\, R_B}{R_A - R_B}$$

Putting $\nu = N_A/N_B = c_A\, R_A/c_B\, R_B$, this can be simplified to

$$c_{A-B} = \frac{c_A\,(\nu - 1)}{\nu - c_A/c_B} \qquad (3)$$

A corresponding formula can be deduced for the constants, c′, of equation (2), assuming that the permeability of the deposit to air flow depends on the surface area of the particles which is related to $\overline{d^2}$.

TABLE 1

CHARACTERISTICS OF THE DUST CLOUDS

Cloud	ν	*N*	Standard deviation of *N*	Diameter (μ) below which proportion of particles was				Mean d^2 (in μ^2)
				20%	50%	80%	95%	
A	—	7,991	9·7	2·27	3·8	6·3	10·4	32·0
B	—	8,021	5·4	2·24	3·6	5·8	8·6	22·5
C	—	4,562	11·0	1·95	3·03	4·7	6·9	15·6
D	—	1,689	15·8	1·38	2·26	3·26	4·6	7·24
C–D	2·70	2,873	—	2·78	3·60	5·3	7·5	19·7
B–D	4·75	6,332	—	2·63	4·02	6·2	8·7	26·3
B–C	1·76	3,459	—	2·90	4·5	6·9	9·4	32·0
A–C	1·75	3,429	—	3·40	5·3	8·6	13·4	55·3
A–D	4·73	6,302	—	2·72	4·3	7·0	11·0	38·0

TABLE 2

SUMMARY OF MEASUREMENTS

Experiment	Cloud	No. of particles per c.cm.			Densitometer		Pressure difference, *P*, in cm. Hg.	*n*	*Nn*
		Cell *a*	Cell *b*	Mean *N*	*i* in microamps.	*R*			
C	A	9,320	8,520	8,875	19·5	26	1·1	5	44,375
E		7,312	7,828	7,570	15·8	36	2·0	10	75,700
F		7,120	6,773	6,947	5·8	90	2·7	20	138,940
G		9,707	7,433	8,570	3·2	138	4·1	25	214,200
				7,991					
Y	B	9,070	8,235	8,652	16·8	33	1·0	10	86,520
Z		7,796	7,621	7,708	12·2	48	1·8	20	154,160
A		7,414	7,652	7,533	7·7	75	2·8	30	225,990
B		8,553	7,827	8,190	4·6	109	3·8	40	327,600
				8,021					
R	C	4,979	4,819	4,899	29·2	11	0·7	10	48,990
W		4,837	4,383	4,610	18·2	29	0·6	20	92,200
S		5,735	4,950	5,343	15·8	36	1·5	40	213,720
X		3,813	3,877	3,845	14·4	40	1·2	40	153,800
T		4,131	3,915	4,023	7·0	81	1·4	80	321,840
U		4,603	2,187	3,395	8·9	67	1·6	80	271,600
V		4,811	3,454	4,123	4·0	120	2·4	150	619,950
				4,562					
I	D	2,202	1,730	1,961	42·2	2	0·1	10	19,610
J		1,426	1,580	1,503	26·3	14·2	0·3	60	90,180
O		1,839	2,074	1,957	23·2	19	0·4	100	195,700
P		1,918	1,331	1,625	23·2	19	0·5	100	162,500
L		1,984	1,622	1,803	18·2	29	0·8	170	306,500
N		1,993	1,975	1,984	15·2	38	0·9	170	337,300
K		1,146	1,252	1,200	10·3	56	1·1	300	375,600
M		1,613	1,336	1,475	10·3	56	1·1	300	400,800
				1,689					

These formulæ are useful because they enable the constants for the densitometer, or the flow resistance increase, to be derived for difference clouds obtained from any two experimental clouds and so extend the information gained from the experiments.

The characteristics of the experimental clouds, A to D, and of the calculated difference clouds are set out in Table 1. The difference in the number of particles per cm.^{3} in clouds A and B is smaller than the experimental error of counting, but the extra particles of A are all large ones and have a considerable effect upon the densitometer and pressure measurements.

Experimental Results

The experimental figures are summarized in Table 2 for the four groups A to D through each of which, as nearly as possible, similar clouds were dispersed.

The quantities c in equation (1) and c′ in equation (2) were computed by least squares from the values of N, n, R, and P given in Table 2. They are shown in Table 3 together with the values for the difference clouds which were computed by means of equation (3). The values of c and c′ relate, therefore, to clouds having different size distributions given in Table 1.

By substituting c and c′ into equations (1) and (2) the deviations between the observations, y'_i, and the calculated values, y_i, were found. These were expressed as percentages of the calculated values and the standard errors,

$$100\sqrt{\frac{\sum_1 [(y'_i - y_i)/y_i]^2}{\text{No. of observations} - 2}},$$

of the experimental results for R and P were worked out. These are also given in Table 3. It will be noticed that the observations are more consistent for the coarser clouds. In fact the pressure readings show so great a scatter as to make this method hardly worth using for fine dust.

TABLE 3

CONSTANTS OF EQUATIONS (1) AND (2), CALCULATED BY LEAST SQUARES FROM THE EXPERIMENTAL DATA AND BY EQUATION (3) FOR THE DIFFERENCE CLOUDS

Cloud	Equation (1)		Equation (2)	
	c	Standard error of experimental values of R from calculated values, %	c'	Standard error of experimental values of P from calculated values, %
A	1,584	18	50,100	31
B	2,970	10	83,500	4
C	4,970	17	227,000	93
D	9,010	25	424,000	44
C–D	3,930	—	179,000	—
B–D	2,520	—	68,800	—
B–C	1,950	—	45,700	—
A–C	800	—	24,600	—
A–D	1,300	—	40,600	—

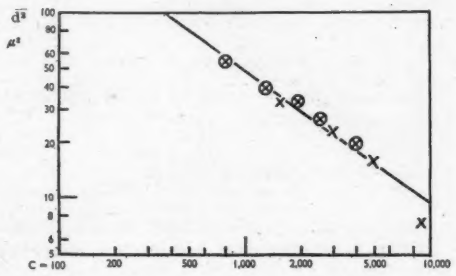

FIGURE.—The relationship between c (equation 1) and the mean of the squares of the particle diameters.

× Experimental clouds A — D.

⊕ Difference clouds calculated by equation 4.

—— Equation 4.

It is clear at once that there is only a small correlation between the constants, c or c′, and the 20 per cent. or 50 per cent. level of the distribution of diameter (Table 1). However, the larger diameters are closely dependent on c and c′.

We see, therefore, that c and c′ depend on the coarse end of the particle size distribution and are not so much upon the finer particles.

The distributions of the squares of the particle diameters have also been calculated and the means of the squares have been tabulated for each cloud. The mean is equal to $\Sigma f_i d_i^2$, where f_i is the proportion or frequency of particles in a small range of sizes centred on d_i and the sums are taken over all sizes present.

If the particles are geometrically similar these distributions relate to the surface area of the particles. In fact, the larger sizes, which include some aggregates, have a greater ratio of surface to mass than the small.

The figure shows the relationship between the constant, c, and the mean of the squares of the diameters. Points for experimental clouds and those calculated for difference clouds fall on a common

curve thus justifying the method used to calculate c_{A-B}. The line plotted is the equation

$$c = 234{,}000\ (\overline{d^3})^{-1\cdot4} \tag{4}$$

All points lie close to this except the one for the finest experimental cloud. Perhaps this is because fine particles are more compact in shape than coarse ones.

The constant of equation (2) for the pressure drop is given by

$$c' = 6{,}680{,}000\ (\overline{d^3})^{-1\cdot4} \tag{5}$$

Discussion

Combining the experimental results leads to the following empirical equation

$$R = 100D^{1\cdot5} = \frac{Nn\ (\overline{d^3})^{1\cdot4}}{234{,}000} \tag{6}$$

This can be used to determine the mean square particle diameter of coal dust if the number concentration, N, is known. Since N is little influenced by a relatively small proportion of coarse particles it can be estimated with the thermal precipitator. For the same reason a sufficiently reliable value for the most probable diameter, d_p, can also be obtained from thermal precipitator counts. From these two quantities, d_p and $\overline{d^3}$, it is possible to construct the entire distribution law if it is of the two-constant type.

For example, coal dust often follows the log-normal distribution,

$$\frac{dN}{d(\ln d)} = \frac{100}{\sqrt{2\pi}\ \ln \sigma_g} \exp\left\{-\tfrac{1}{2}\left[\frac{\ln (d/d_m)}{\ln \sigma_g}\right]^2\right\} \tag{7}$$

where d_m is the median diameter and $\ln \sigma_g$ is the standard deviation of the logarithms of the diameters. ln signifies the natural logarithm.

In this case the median can be found from the experimental observations using

$$d_m^4 = \overline{d^3} \cdot d_p^3 \tag{8}$$

and the other constant from

$$(\ln \sigma_g)^2 = \ln (d_m/d_p) \tag{9}$$

These expressions were deduced from equation (7).

It is thus possible to use the thermal precipitator, which is not satisfactory for coarse dust, in conjunction with the P.R.U. hand-pump outfit, which is insensitive to fine dust, as a means of obtaining the complete size distribution of airborne coal dust.

The pressure difference method of assessing deposits on filter paper is not, at present, recommended.

Summary

Airborne dust is frequently assessed by aspiration through a filter paper followed by photoelectric measurement of the resulting stain. This account of experiments with coal dust shows how the calibration depends on particle size, and the low sensitivity of the method for small particles, when large ones are also present, is exposed. The work was carried out with a hand-pump and densitometer developed by the Pneumoconiosis Research Unit of the Medical Research Council. The theoretical basis of the measurement is discussed. The increase in resistance to air flow through the filter paper, due to the deposition of coal dust, was also studied.

The authors are grateful to Mr. P. Armitage for his careful criticism of this paper, and to Dr. T. Bedford for his interest and encouragement while the work was in progress.

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BRITISH AND FOREIGN OFFICIAL PUBLICATIONS

Medical X-Ray Protection up to Two Million Volts. Handbook 41, U.S. Department of Commerce, National Bureau of Standards, 1949. Pp. vi+43; six figures. Price 15 cents.

This little handbook is one of a series prepared or being prepared by a number of subcommittees drawn from the U.S.A. National Committee on Radiation Protection and sponsored by the National Bureau of Standards. The importance of the information and expert guidance contained in this issue is out of all proportion to its size.

It is inevitable that even the safety aspects of radiation should at the present time be permeated with conceptions and measurements of a highly technical kind. Ultimately, it may be presumed, the maintained application of radiant energy or atomic energy to peaceful ends will render possible so wide a dissemination of knowledge on the practical fundamentals of the subject that workers and technicians will be able to handle the external problems without any knowledge of the existence of Planck's constant as effectively as an engine-driver does an engine without any necessary appreciation of the second law of thermodynamics.

In the present stage of development it is obvious that codes of safety rules for the protection of workers cannot be couched in absolutely non-technical language, nor can the responsibility for safe working be placed on anybody other than well-trained experts. In fact the little handbook, in its excellent introductory definition, describes such an expert thus: "A person having the knowledge and training needed to measure radiations and to advise regarding radiation hazards. The physician or dentist employing him shall be responsible for seeing that he has the necessary training and education. Certification by competent authority by a board set up for the purpose is desirable."

Although it is claimed that the "recommendations are intended primarily for the protection of the radiation worker and not for the patient," in fact considerable guidance is given in the safe practice of radiation therapy from the patient's point of view. It will be known, even to non-radiologists, that there has been a good deal of difference of opinion as to the maximum total dose of radiation of the hazardous kinds that is permissible for the human subject, whether a patient treated with a primary radiation passing through a controlled aperture, or a worker exposed to direct radiation from the source, to secondary radiation arising from the surrounding matter struck by the primary radiation, or to scattered and stray radiation.

The International *X*-ray and Radium Protection Commission at the Fifth International Congress of Radiology in 1937, and the British *X*-ray and Radium Protection Committee's revised recommendations of 1938, led to the more or less general acceptance of 1 international Röntgen (r) per week as the tolerance limit for normal persons working in satisfactory conditions. This limit was accepted by, among others, the Australian Department of Health, which on the basis of a seven-hour-a-day continuous radiation and a five-day week laid down as the toleration dosage approximately 10^{-5}r per second. The precise reason for setting this limit is by no means clear.

The Atomic Sciences Committee of the Association of Scientific Workers in 1948 issued a pamphlet entitled "Protection of Workers from Radiation Hazards" and indicated that 0·1 r per eight-hour day and 0·5 r per five-day week was a generally accepted tolerance-dose. But, while it was pointed out that this level was at that time accepted in the U.S.A., it was added that future work might tend towards lowering this figure, and that all effort should be made to keep below the so-called permissible maximum of that time.

As in all cases in which it is necessary to lay down maximum permissible concentrations—whether of gases, dusts, fumes, or radiations—the problem is in part to find the maximum concentration which will not adversely affect, acutely or chronically, any system or organ in the body. Thus, in the case of x rays and γ rays, it is quite conceivable that a maximum dose could be laid down which would not manifestly harm any organ system and which yet would in the long run exercise severe genetic effects on succeeding generations of descendants of the exposed persons.

Considerations of this kind, we imagine, have led the compilers of this little handbook to drop the maximum permissible dosage rate to 0·3 r per week or, based upon a forty-eight-hour week, $0{\cdot}17\times10^{-5}\frac{\text{r}}{\text{sec.}}$, which is about $\frac{1}{6}$ of the dose per unit time of the 1938 standard.

This is an important revision for it is manifestly less pessimistic than that of the International Commission on *X*-ray and Radium Protection which brought down the tolerance dose to 0·02 r per day, which (as stated by L. F. Curtiss in Patty's "Industrial Hygiene and Toxicology," 1948) was "intended to take care of genetic injuries and may be subject to further revision as additional information on this subject becomes available." Having regard to the fact that L. F. Curtiss served in the preparation of the handbook (issued in 1949) here reviewed, and was responsible for the statement quoted, it is apparent that second thoughts may appear overnight in this matter.

It is interesting to note that Koelsch, in his revised *Lehrbuch der Arbeitshygiene* issued as late as 1947, still gives the tolerance dose as 0·25 r *per day* and 1·25 r per week, but discretion was given by a German Government decree in 1941 to the Factory Inspectorate to forbid the employment of certain age-groups of workers if the daily dose exceeded 0·025 r.

It seems reasonable to conclude that even 0·3 r per week may not be a final agreed maximum: much must yet remain to be discovered on perhaps subtler actions of the penetrating rays, and modifications may still be necessary. Judged from the results of the well-known experiments on *Drosophila melanogaster*, it must be kept in mind that the rise in mutation rate brought about by radiation is proportional to total dose, so that a small dose repeated over many years will have the same genetic effect as a large dose given on a single occasion.

Still, it has to be remembered that mutations are being produced in nature all the time, that defective genes in the enormous majority of cases fail in the process of mitosis, and that the likelihood of two defective genes meeting is significant only when both parties have worked in radiations. In medical radiological practice it requires a good deal of careless ignorance to produce even temporary sterility in males (300 to 700 r) and permanent damage in female technicians having regard to the significant protection layer between the surface and the ovaries.

Maximum dosage at 0·3 r per week should be quite safe hæmatologically: radiologists in this country have found that even 0·17 r per day for twenty months produced no effect, clinically or hæmatologically. As for effects on the skin, it is probably true to say that one Röntgen per day (1 r) can be tolerated by the normal skin indefinitely without damage. Still, it must be recorded that some American workers consider that 0·1 r per day is a safer maximum to work to for the skin.

From the industrial medicine point of view the recommendations include some interesting points. Pre-employment examination is essential and must include an occupational history, a description of any unusual radiation exposure from previous examinations, diagnostic radiographic examinations or any radiation therapy, a detailed family history for hereditable disease, a complete physical examination, *x*-ray examination of the chest, and a complete blood count (including platelets). In the case of childless married persons, a record must be kept as to whether childlessness was due to birth-control measures. It is recommended that blood counts should be performed at 3 p.m. on two or three successive days to indicate in some measure the variations characteristic of the individual. A total white count of less than 4,000 should lead to rejection. Unless monitoring of installations and personnel (pocket ionization chambers or films) is practised, blood counts should be made every three months, otherwise not less frequently than every six months. A fall in white counts of 2,000 or more may indicate injury and should be investigated, but such a fall is often a normal variation. Notable authority has sometimes been scathing on the value of blood counts: reliable quantitative deviations from "normal" as an index of *early* radiation disease are not yet available.

In addition there should be a routine annual physical examination and a review of the medical history for all workers exposed to radiation as a routine.

A significant omission in the health precautions is any recommendation as to the length of holiday to be given and the maximum time to be worked in a single day. The German decree of 1941 laid down that workers in non-medical radiation and radio-active work must be given a minimum of eighteen days' holiday, of which fourteen days must be consecutive. The same decree states that not more than eight hours must be worked in one day under any circumstances, and not more than forty-eight hours a week, but, further, if more than twenty-four hours per week are worked, for every additional two hours worked one hour's free time on full pay must be allowed by deduction from the working time in the same week, and no other work given in that free time. This signifies that a forty-eight hour week on this kind of work is really thirty-six hours' actual work and twelve hours' free time which, the decree says, should as far as possible be consecutive. The American maximum of 0·3 r probably is low enough to obviate the necessity of a regulation such as that in the German decree, but there can be no doubt of the need, or at least desirability, of a holiday interval of at least fourteen consecutive days per year in addition to the monitoring control which may determine longer periods of freedom from all exposure.

Many of the sharply indicated precautions in this handbook deserve appreciative comment. We wonder how frequently in dental *x*-ray work in this country the following precaution is practised: "Under no condition shall the operator or any regular assistant hold a film in place when an *x* ray is taken. In case of emergency a relative or other person not habitually exposed to *x* rays may hold the film." (Precaution for Dental Roentgen Installation operated at Potentials up to 70 Kv peak.) Or in hospital radiographic departments, how often the following rule is infringed: "No persons other than the patient and those needed to hold the patient shall be in the radiographic room during exposure."

Very valuable is the reminder that "grenz rays" (soft *x* rays produced at less than 15 Kv) "may cause the same kind of injurious effects as harder *x* rays, although limited to superficial layers of tissue."

Whilst it is reasonable to assume nowadays that clinicians do not engage in the long conversations they were wont to enjoy in the *x*-ray departments, it may yet be wise to apply the recommendation by the authors of the handbook that where therapeutic work is going on at potentials over 250 Kv there should be a notice such as "*X-rays*! *Do not loiter*" at all infrequently occupied spaces where radiation hazards exist, and it is desirable to have such notices illuminated. It seems almost redundant to include among the recommendations on electrical protection one forbidding installation or operation of *x*-ray apparatus in dangerous locations, such as an anæsthesia room, unless it is explosion proof; but evidently the authors prefer to be safe rather than sorry.

The severely practical nature of this handbook has obviously necessitated a great deal of compression and a minimum of theoretical treatment. Valuable curves of radiation attenuation for potentials from 75 Kvp to 2,000 Kvp are given in thickness of Pb and concrete. Tables giving primary protection-barrier requirements (in Pb or concrete as necessary) for potentials from 400 Kvp to 2,000 Kvp for different target currents, and target distances enable calculations to be made for barrier thicknesses required as protection at occupied points around the tubes.

Although this handbook is meant for operators of medical *x*-ray apparatus, it can be read with some pleasure if not with ease by anyone who understands a little about medical radiography and desires to appreciate the great strides being made towards safety in this specialized and potentially very dangerous field.

M. W. Goldblatt.

BOOK REVIEW

Welfare in Industry. Edited by Blayney Thomas. Published by the Caxton Publishing Co. Ltd. 1949. Pp. 590 ; illustrated. Price 47*s.* 6*d.*

This book has been " planned by a group of people in an attempt to equip the newly appointed welfare officer with a readily available guide to his work " and to provide a symposium for those already experienced in welfare work in industry. The Editor, Blayney Thomas, and his thirteen collaborators are all recognized authorities in their particular spheres of welfare in British industry.

The contributors cover the whole field of welfare in industry ranging from the historical survey of the industrial revolution up to the modern conceptions in health, welfare, safety, and physical environment in industry. The work of the welfare officer in industry is dealt with fully, and also the plans for his day-to-day work, the liaisons which are so important to good welfare, and the background and qualifications required of the industrial welfare officer. Throughout the whole book emphasis is laid upon the basic, human desiderata of those who are responsible for dealing with many of the urgent human problems in industry today. The chapters on recruitment and induction of the new employee, of education and training, are perhaps as helpful as any which have been published in the literature on the subject. They are essentially practical, and stress the value of films and other visual aids in training, the importance of magazines, and general educational propaganda as a means of integrating industrial society and of adding to the general standard of education and all who work within the factory. The chapters on health and hygiene written by two well-known experts present to the new welfare officer their particular specialities in a helpful way. Similarly, the chapter on canteens, their design, equipment, and planning, cover this vast and important subject fully, and the information contained in this chapter is of great value. The last chapters of the book deal with the problems of accident prevention and the newer developments in the use of colour and lighting as part of the physical and psychological environment in industry.

The Editor and his collaborators have succeeded in their task. This book is probably the most comprehensive textbook on welfare in industry published in Great Britain. The references given throughout to outside social institutions, Government bodies, and publications are very helpful. The type is easy to read, and the illustrations and plans are well produced. The Editor is to be congratulated upon having brought together so much valuable information concerning those many facets of human activity within the field of industry which we know under the generic term " welfare."

A. J. A.

Owing to the steadily increasing cost of production, it has been found necessary to raise the price of the *British Journal of Industrial Medicine*, beginning with the first issue of 1950. The yearly subscription will then be 30*s.* (U.S.A. $5). Single copies remain at 7*s.* 6*d.* each.

ABSTRACTS

(This section of the JOURNAL *is published in collaboration with the two abstracting Journals, Abstracts of World Medicine, and Abstracts of World Surgery, Obstetrics, and Gynæcology, published by the British Medical Association. The abstracts are divided into the following sections : toxicology ; industrial physiology ; industrial lung disease ; industrial skin diseases ; accidents and orthopædic surgery ; industrial ophthalmology ; environment ; general. Not all sections will necessarily be represented in any one issue)*

TOXICOLOGY

The Effect of Dimercaprol (B.A.L.) in Acute Iron Poisoning. EDGE, N. D., and SOMERS, G. F. (1948). *Quart. J. Pharm.*, **21**, 364.

The effect of 2 : 3-dimercaptopropanol (BAL) in acute experimental iron poisoning was investigated. When iron was given orally, either as the sulphate or as the chloride, it was found that BAL afforded a slight protective action against ferrous sulphate if administered 15 minutes before the iron salt, but made the animals slightly more susceptible if given after ferrous sulphate or ferrous chloride. The animals used were mice, the MLD being as follows : for ferrous sulphate (as Fe) 710 mg. per kg. ; when preceded by BAL (100 mg. per kg. given orally) 1,050 mg. per kg. ; when followed by BAL 15 minutes after the iron, 440 mg. per kg. The corresponding figures for ferrous chloride (as Fe) were 840 mg., 594 mg., and 340 mg. per kg. When BAL and the iron salts were administered intravenously it was found that BAL significantly increased the toxicity of iron, even though the drug was given before the iron ; the MLD for ferrous sulphate (as Fe intravenous) was 13·8 mg. per kg. With previous injection of BAL the MLD was 8·1 mg. per kg. The corresponding figures for ferrous chloride were 18·5 mg. per kg. and 10·2 mg. per kg.

A BAL-iron complex was prepared and injected intravenously. Its toxicity was greater than that of the corresponding amount of iron, and the MLD corresponded to that of an iron salt preceded by BAL. *J. D. Judah.*

Pathological Anatomy of Acute Experimental Beryllium Poisoning. SCOTT, J. K. (1948). *Arch. Path.*, **45**, 354.

The effects of beryllium sulphate on rats, rabbits, and guinea-pigs, are described.

The lethal dose varied with the species, but for white female rats weighing 175 to 200 g. the LD 50 was about 7·2 mg. per kilo. Petechial hæmorrhages and serous effusions occurred when large doses were used. All rats dying more than 3 days after a single intravenous dose developed jaundice. Liver necrosis varying for small mid-zonal foci to necrosis of almost the entire organ was observed. The extent of necrosis and time interval required depended on the dose. The necrotic cells within 2 to 3 days underwent liquefaction and absorption, when phagocytes, granulocytes, and lymphocytes appeared. After a single injection of beryllium, regeneration began on the fifth or sixth day and was complete within 2 weeks, leaving no scar. A smaller dose of beryllium was sufficient to produce renal damage which took the form of necrosis of the epithelium of the distal part of the first convoluted tubule. The most severe lesions were seen in rabbits which had inhaled beryllium sulphate dust. The spleen became enlarged and congested and the number of nucleated cells decreased. Nuclear degeneration was seen in the spleen, lymph nodes, and bone marrow, although smaller doses produced granulocytic hyperplasia.

Local effects were produced by inhalation of the dust for 6 hours daily for 11 days, the concentration used being about 88 mg. per cubic metre of air. Guinea-pigs and dogs showed severe acute conjunctivitis while some animals developed keratitis, some with ulceration and hypopyon. In all animals, an inflammatory exudate was found in the terminal bronchi together with desquamation of the epithelium. The exudate became coagulated and contained nuclear debris, and a few phagocytes were seen in the alveoli. Granulocytes were scarce. Although occasional fibroblasts were seen growing into the exudate, pulmonary fibrosis was never observed.

R. B. T. Baldwin.

Environmental Studies in Plants and Laboratories Using Beryllium : The Acute Disease. EISENBUD, M., BERGHOUT, C. F., and STEADMAN, L. T. (1948). *J. industr. Hyg.*, **30**, 281.

Cases of acute beryllium poisoning have been reported from four industrial processes in the United States. The present investigation was carried out in three plants producing beryllium compounds from the ore, and in a number of laboratories and shops engaged in research on the ceramics and metallurgy of beryllium. Over 500 air samples were collected on filter paper and analysed spectrographically. The operations involving beryllium sulphate and beryllium fluoride are associated with a high incidence of pneumonitis, the total of beryllium inhaled daily averaging about 0·85 mg. and 0·55 mg. respectively. The evidence obtained with beryllium oxide is contradictory, for several severe cases of poisoning resulted from a daily inhalation of 4 mg., while in another operation, where the daily inhalation was 300 mg., no cases were reported. There were no cases of poisoning from inhalation of 13 mg. of beryl ore, but there were 2 mild cases when 1 mg. of pure beryllium dust was inhaled.

The authors studied 3 cases of acute pneumonitis and 5 of bronchitis which followed relatively short exposures to high concentrations of the fluoride and sulphate. In one case the furnace in a big room suddenly discharged a large volume of fumes containing beryllium fluoride. Air samples were collected 10 minutes later, and it was calculated that about 45 μg. of beryllium were inhaled by each of the 8 employees exposed. One developed pneumonitis and 2 bronchitis. *H. M. Vernon.*

The Determination of Thallium in Urine. ACKERMAN, H. H. (1948). *J. industr. Hyg.*, **30**, 300.

The method described here for the determination of micro quantities of thallium in urine is a modification of the methods used by Shaw and Haddock.

Modes of Penetration of Mercury into, and its Removal from Protective Clothing. (In Russian.) PYANKOV, V. A. (1948). *Gigiena*, 8, 19.

Preliminary investigations at a large mercury plant showed that mercury can penetrate into protective clothing: (*a*) by being absorbed by the fibres of the material from which the clothing is made or by the dyestuff used in the dyeing of such clothing; (*b*) by impregnating, in the form of minute droplets, the interstices of the textile fabric of clothing. Tests showed that the samples of fabric dyed black and blue absorbed greater amounts of mercury than similar samples of another colour or those undyed. It was found that the increased absorption of mercury by the black cotton fabric or by the dark-blue woollen cloth was due to the dyestuff; a square decimetre of black fabric weighing 2·9 g. kept in an exsiccator over metallic mercury for a month at 25° C. absorbed 334 μg. but a similar sample after decoloration by calcium chloride absorbed under the same conditions only 4·3 μg. of mercury. Absorption of mercury vapour by clothing was in direct ratio to mercury concentration in the air and to the time of exposure. In four samples of textiles impregnated with mercury vapour and stored in the open air at 25° C. for a month the average decrease of mercury concentration in the fabric was 70 to 75%, of which 50% was eliminated in the first 20 hours of storage. The rate of evaporation of mercury adsorbed on the fabric was directly proportional to the radius of mercury droplets impregnating the fabric. By heating at 100° C. for 2 hours, mercury impregnating the textile material was eliminated. *H. P. Fox.*

Experimental Intoxication with Selenium. (Intossicazione sperimentale da selenio.) VESCE, C. A. (1948). *Folia med., Napoli*, **31**, 415.

The halides of selenium and also SeO_2, SeO_3, H_2SeO_3 and H_2SeO_4 are particularly dangerous to man, either by inhalation of dust and vapours or by cutaneous absorption. In industrial cases poisoning is generally acute whereas in endemic areas the effects are gradual. Irritation of the skin and mucosæ, loss of the sense of smell, and irritation of the respiratory and alimentary tract have frequently been observed. Physical injury is generalized. The author is interested in the action on the nervous system and the endocrines. Degenerative changes are recorded in the liver and kidney, and in the reticuloendothelial system of the spleen hypertrophic changes have been observed. Experiments were made on 5 dogs and 5 guinea-pigs, which were given sodium selenide by mouth. The former received a solution of 1 mg. in 20 ml. of water and the latter 1 mg. in 20 ml., dosage varying with weight and 0·5 mg. being given per kilo on the first day; this dose was increased in stages. Some of the animals were finally killed, others died between the 40th and 70th days. Progressive asthenia, loss of weight and appetite, vomiting, and occasional irritability were noted before death.

At necropsy on the guinea-pigs and dogs some pulmonary infarcts were seen. The hearts of the guinea-pigs were of abnormal colour and texture, but in the dogs no cardiac changes were seen. Three out of the 5 guinea-pigs had ulcers on the smaller curvature of the stomach, but the dogs were not thus affected. In all the animals some inflammation of the remaining alimentary tract was seen. Fatty degeneration and cloudy swelling were seen in the liver of the guinea-pigs and in the dogs the organ was enlarged and more firm than usual, while the connective tissue was increased. Some congestive or degenerative changes were seen in the spleen and kidneys of most of the animals. The microscopical changes tallied with the macroscopical and agree with those described by Dudley. *G. C. Pether.*

Methyl Chloride Intoxication. (L'intoxication par le chlorure de méthyle.) ROCHE, L., and BOUCHET, J. (1948). *Arch. Mal. prof.*, **9**, 406.

Methyl chloride poisoning generally develops after a few hours and the free interval is of diagnostic importance. Digestive symptoms are usual and include nausea, vomiting, and intestinal disturbance such as diarrhœa. Vertigo, somnolence and ataxia may be observed; headaches are common. Convulsions and epileptiform attacks generally precede or accompany coma. Asthenia is not uncommon after mild poisoning. In moderate poisoning there is somnolence, insomnia, and headache or confusion. There may be visual disturbance with diplopia or difficulty of accommodation. The gait may be uncertain and the tendon reflexes brisk without other signs of pyramidal involvement. If coma develops the prognosis is grave; fever and albuminuria preceding anuria may presage death. Some forms of poisoning may resemble epidemic encephalitis with hiccup, drowsiness, choreiform movements, and visual disturbance.

Foodstuffs kept in cold chambers in which methyl chloride is the freezing agent may be accidentally contaminated. If exposure to fumes is intermittent it may not be dangerous provided that periods of non-exposure are sufficient and regular. It is thought that the nervous symptoms are due to a capillary vasodilatation and general vasomotor disturbance. Some authors suggest that the compound breaks up and that free methyl alcohol is liberated, but no damage to the ocular nerve has been observed.

The odour of methyl chloride is slight. A special lamp may be used for detection, since the vapour burns with a bluish-green colour. Normally the lamp burns with a pale bluish colour but methyl chloride vapour makes the flame more blue or blue-green. Safety engineers sometimes add more pungent substances to methyl chloride as a precaution, or irritants such as acroleine. Treatment of poisoning is symptomatic, with oxygen, alkalis, and cardiac stimulants; dehydration should receive special attention. If sedatives are needed potassium bromide is advised but never chloral or bromoform. *G. C. Pether.*

Methyl Bromide Poisoning. (Les intoxications par le bromure de méthyle.) ROGER, H., and SIMONIN, R. (1948). *Ann. Méd.*, **49**, 486.

The authors give a full clinical description of methyl bromide poisoning. In mild cases there is vertigo, drowsiness, and headache, with return to normal within a few days. In cases of average intensity, the above symptoms are more severe and there may be tremor and myoclonic jerks. There is marked asthenia, and painful hyperacusis and transient blindness occasionally occur. All these symptoms take some 3 months to clear up. In severe poisoning, symptoms appear either immediately or after a latent period of 2 to 3 hours. Death may occur within 48 hours in status epilepticus. In other cases the patient recovers from coma but continues to have fits. A peculiar tremor develops, usually unilateral and affecting mainly the arms. There may be other signs of extrapyramidal involvement. All these disturbances are permanent. Less conspicuous are mental changes, signs of cerebello-labyrinthine involvement, and visual disturbances. The authors believe that the

condition may be accounted for by a vasomotor crisis leading to more or less permanent lesions.

J. F. Delafresnaye.

Cardiac Stimulation in Carbon Monoxide Asphyxia. SCHWERMA, H., IVY, A. C., and FRIEDMAN, H. (1948). *J. appl. Physiol.*, **1**, 364.

Since death from carbon monoxide asphyxia may be primarily due to cardiovascular failure the authors investigated the effects of cardiac stimulants as an adjunct to other methods of resuscitation, 45 dogs being treated with 0·3% carbon monoxide till the onset of spasmodic gasps of air hunger ; 1 minute after the last gasp treatment was begun. All dogs received 100% oxygen, and were placed in 3 groups : (A) receiving no other treatment ; (B) receiving 1 mg. adrenaline hydrochloride and 0·65 mg. atropine sulphate in 1 ml. normal saline, injected into the heart ; (C) given 1 ml. normal saline into the heart. Each group consisted of 15 dogs.

No dog in group A or C survived. Of those in group B, 2 remained comatose till death after 68 and 16 hours ; a third died in 36 hours and one survived and was apparently normal 30 days after the experiment. Transient cardiac activity was induced by the needle and normal saline in group C but this was insufficient to maintain the heart beat. The authors conclude that it is probably worth while trying intracardiac adrenaline and atropine as a last resort in cases of carbon monoxide poisoning in humans. As adrenaline shows parasympathetic as well as sympathetic activity on the heart in asphyxia atropine is added to neutralize the former effect. (Danielopolu and Marcou, *Pr. méd.*, 1940, **48**, 44).

A. T. Macqueen.

Elimination of Carbon Monoxide from the Blood of Acutely Poisoned Dogs. SCHWERMA, H., WOLMAN, W., SIDWELL, A. E., and IVY, A. C. (1948). *J. appl. Physiol.*, **1**, 350.

The authors investigated the rate of clearance of carbon monoxide from dogs resuscitated by various methods. The dogs were exposed to air flowing at a constant rate and containing 0·3% carbon monoxide ; at the first sign of air hunger they were removed from the chamber and treatment began. As it was impossible to keep the resuscitator masks on the dogs after they became conscious, the studies are based on 2 to 4 minutes of treatment followed by a period of 4 hours during which the animal inhaled air.

The dogs were divided into the following 4 groups : (I) Eight exposed to carbon monoxide till death. Average saturation of blood with the gas was 72·8%. (II) 20 dogs removed from the chamber at the first gasp of air hunger, and which failed to survive ; blood saturation averaged 73·2%. (III) 41 dogs successfully revived after removal at the first gasp of air hunger ; saturation averaged 74·3%. (IV) 7 dogs in which resuscitation failed ; in these the blood from spleen was 77·5% saturated, from liver 79·1% saturated, and from the portal vein 68·4% saturated. The differences between hæmoglobin concentrations in survivors and non-survivors were not significant. The individual variations were considerable and inexplicable. At necropsy the spleen was found to be hard and contracted and the liver engorged. The latter organ may be a source of carbonyl-hæmoglobin after respiration has started, owing to its large capacity for blood storage.

The authors compare the findings of others with their own. The rate of clearance of carbon monoxide from the blood was the same during the first 5 minutes with all methods in which oxygen was used, but the initial rapid rate persisted for a further 10 minutes with the carbon dioxide and oxygen mixture. Four hours after treatment the blood carbon monoxide level was approximately the same in the blood of all dogs. Mechanical artificial respiration with either pure oxygen or a 7% carbon dioxide and oxygen mixture was the most effective method ; it is considered that there is no advantage in using carbon dioxide. In any case good cardiac function is essential to satisfactory delivery of oxygen to the tissues. It is claimed that by raising the oxygen (in physical solution) level in plasma from 0·5% to 2·5% by the use of pure oxygen the dissociation of carbonyl-hæmoglobin is accelerated and oxygen made available to the tissues in a form in which it can be most readily taken up. Furthermore, the first organ to receive the improved oxygen supply will be the heart itself, a further contribution to increased cardiac output and recovery.

A. T. Macqueen.

Dietary Protein and Carbon Tetrachloride Intoxication. Histological Changes in the Rat's Liver in Acute Poisoning. (In English.) LAQUEUR, W., and NASHAT, F. (1948). *Arch. int. Pharmacodyn.*, **77**, 449.

Adult rats were given single injections of carbon tetrachloride (0·01 ml. per 100 g. body weight) after being kept for 3 weeks on 20% and 4% casein diets. The severity, extent, and rate of healing of the liver lesions were compared over 7 days. With adequate protein in the diet (20% casein) lesions were zonal, but with a low protein (4% casein) intake such lesions tended to become more diffuse. Females reacted more severely than males. The authors think that these differences are due to failure or impairment of detoxication within the liver cells consequent on reduced protein.

G. R. Cameron.

Industrial Poisoning with Nitrogen Mustard. (Gewerbliche Vergiftungen durch Beta-Beta'-Dichlordiäthylmethylamin (Stickstoff-Yperit, Nitrogen Mustard).) JORDI, A. (1948). *Helv. med. Acta*, **15**, 470.

The author describes 7 cases of poisoning by nitrogen mustard. Lesions are almost identical with those which develop from exposure to the sulphur analogue. Blisters developed more slowly than with the latter substance, in one case after 8 days. The connective tissue round the eyes and the upper respiratory tract was readily affected, and hoarseness was often prolonged. In one patient toxic pneumonia developed.

Nitrogen mustard is damaging because of its effects on enzymes. Increased urobilinogen appeared early in the urine, and all the 5 patients investigated had hepatic involvement. Bilirubin was sometimes increased in the blood, with a positive Takata-Ara reaction. General lassitude was a common symptom. Changes of pigmentation were seen similar to those occurring with mustard gas. Some patients had a yellowish coloration of the conjunctiva, others had urticarial spots, œdema of the lips, or night blindness. In the skin and mucous membranes an allergic state develops to the irritant as with mustard gas. Bronchial asthma may develop after prolonged inhalation of α, β-dichlorodiethyl ether, but in contrast the α, α'-dichloroethyl exerts only a general toxic effect.

The blisters caused by nitrogen mustard were treated by application of 1 in 3,000 silver nitrate, the chlorine ion being rapidly fixed by the silver. Cod-liver oil ointment has also been useful in treating wounds and conjunctivitis.

The general intoxication may be partly remedied by injections of vitamins including C and B_1. Injections of a calcium preparation appeared to benefit the respiratory symptoms, and the unsightly pigmentations of the skin responded to the application of 10% salol in " vaseline."

Prophylaxis is important, and the author does not think that nitrogen mustard has such a selective action as to be free from risk when used therapeutically. Although the workers examined were in fact exposed very little, it appears that more severe contamination would have caused permanent damage. Nitrogen mustard should be listed as a toxic substance so that victims of industrial poisoning may be compensated.

G. C. Pether.

Possibility of Replacement of Aromatic Hydrocarbons by Less Toxic Products. (Possibilités de remplacement des hydrocarbures benzéniques par des produits moins nocifs.) LAFARGE, P. (1948). *Arch. Mal. prof.*, **9**, 378.

A commission was recently appointed to discover safe substitutes for aromatic hydrocarbons.

It is concluded that no general prohibition of the benzene hydrocarbons is possible. Trichlorethylene has many advantages, though some workers dislike it despite medical recommendation. The petroleum solvents are generally complex substances and successive samples of what are alleged to be the same substance are found to vary considerably. It would seem desirable to make exclusive use of Venezuelan oils for cleansing purposes, since these contain low percentages of aromatic compounds. In many cases light solvent naphthas could be substituted for benzene. Benzene should be forbidden and both toluenes and xylenes should be defined and their use controlled. *G. C. Pether.*

Study of Punctate Basophilia in the Accumulator Industry. (Étude de la granulobasophilie hématique dans l'industrie des accumulateurs.) DUVOIR, M., DEROBERT, L., and HADENGUE, A. (1948). *Arch. Mal. prof.*, **9**, 365.

Investigations were carried out in an accumulator factory to determine the processes involving most risk. It was noted that some production of lead dust occurred in the foundry when plates were being trimmed and that greater risks were present in the process of coating these, especially if this was done by hand rather than by machine. Material shortages made it necessary to recover used plates, a particularly dangerous procedure.

Among 50 workers 38 never had punctate basophilia. These men worked in the foundry; the other 12 had slight and transient changes. Six of the 50 men had anæmia with a red cell count between 3 and 4 million per c.mm. of blood. The great danger of the kneading process was countered by giving the men 3 alternate weeks in completely safe employment. Masks were worn and the shop was kept damp. Blood examinations were made every 2 months. Three-fifths of the men had basophil changes. In the coating and assembly shops 13 out of 38 men never had pathological blood changes; 12 had transient and 13 permanent variations from normal. The personnel working on the recovery of old batteries was frequently changed so that only 15 men could be examined at frequent intervals; 40% had permanent basophilia. Some sweepers and cleaners also had blood changes: they took no precautions and were variously employed.

Basophilia generally develops, if at all, in the first or second month of employment. In 1 case it was noted after 8 days. It appears desirable to cease exposure of any men with more than 0·5 basophilic cells per field, but economic necessity made it unavoidable to employ some men after the basophilia had reached a theoretically dangerous level.

Basophilia rapidly diminishes when the workman is removed from any lead hazard and generally disappears in 2 or 3 months but may persist in moderate form for a year. It was not possible to determine the relation between the degree of basophilia and the level of lead in the blood. *G. C. Pether.*

A Tropical Occupational Disease due to Derris, with Investigations of the Effect of Derris on Goldfish. (Eine tropische Berufskrankheit durch Derris mit Untersuchungen über die Wirkung von Derris auf Goldfische.) SIMONS, R. D. G. P. (1948). *Acta derm.-venereol., Stockh.*, **28**, 601.

The bark of the derris root contains rotenone, degueline, tephrosine, and toxicarol. In experiments with goldfish, derris powder was found to exert a stronger lethal action than did rotenone. From the literature some fatal cases of derris poisoning are described. The symptomatology of less severe poisoning is illustrated by a number of cases occurring among the workers in a derris factory in Java. The symptoms include dermatitis and irritation of the mucous membranes—rhinitis, conjunctivitis, pharyngitis, laryngitis, and balanoposthitis. Pruritus ani, loss of taste and appetite, nausea and vomiting, and abdominal pain may occur. The author cautions against the use of derris in medical preparations as, apart from their possible toxic effects, he believes that their medical value is doubtful.

G. W. Csonka.

Comparative Chronic Toxicity for Warm-blooded Animals of 2,2-bis-(*p*-chlorophenyl)-1,1,1-trichloroethane (DDT) and 2,2-bis-(*p*-chlorophenyl)-1,1-dichloroethane (DDD). HAAG, H. B., FINNEGAN, J. K., LARSON, P. S., DREYFUSS, M. L., MAIN, R. J., and RIESE, W. (1948). *Industr. Med.*, **17**, 477.

The relative toxicities of DDD and DDT were investigated by feeding 119 rats, including controls, with the drugs for periods of up to one year, by skin absorption tests involving patch inunction or immersion of 30 rabbits in xylene and triton emulsions and dimethyl phthalate solution, and by inhalation of (*a*) dust in 12 dogs, 12 rabbits, and 60 rats, (*b*) " ultrasene " sprays in 18 dogs and 80 rats, and (*c*) emulsion sprays in 9 dogs and 50 rats. The toxicity was assessed by the appearance of tremors, the effect on growth, the mortality rate, and the histopathology of the liver. DDD is found to be one-third as toxic as DDT on prolonged feeding and one-quarter as toxic by skin absorption to rabbits; it is much less toxic to rats by dust inhalation. DDD is more toxic than DDT to rats by ultrasene spray inhalation, but less toxic by emulsion spray. The first visible sign of chronic DDT poisoning is tremor; that of DDD is diminished rate of growth, a sign which appeared at levels much below the lethal, tremor not being observed. The most characteristic lesions produced by DDD were fatty degeneration and miliary necroses of the liver. Neither DDD nor DDT caused primary irritation or sensitization in patch tests in man.

W. E. Kershaw.

INDUSTRIAL PHYSIOLOGY

Factors Influencing Resistance to Cold Environments. LANGE, K., GOLD, M. M. A., WEINER, D., and KRAMER, M. (1948). *U.S. Army med Dept.*, **8**, 849.

Rabbits were exposed to a temperature of −20° C. and the skin and rectal temperatures were recorded until the animals died. The survival time was markedly decreased by restriction of movement. The body temperature of animals so restricted fell steadily from the start of the exposure, while that of the control animals was maintained until shortly before death, when it fell rapidly. Light anæsthesia or alcoholic intoxication decreased survival time. When thyroid extract was given for several days before exposure survival time increased; administration of thiouracil shortened survival time. The results are discussed in relation to the survival of military personnel exposed to Arctic conditions.

R. A. Gregory.

Death from Cold. UTTLEY, K. F. M. (1948). *N.Z. med. J.*, **47**, 427.

Three girls died from cold while on a walking tour in the New Zealand hills. Post-mortem examinations were made 60 hours after death. There was pinkish mottling of the legs and forearms, with pronounced swelling of the parts, which were doughy but did not pit on pressure. The fat was very firm. In each pericardial cavity there was 100 ml. of serous fluid. The blood was brighter red than usual. There were numerous solid dark red masses, ¾ inch to 1½ inches in diameter, in the lungs. Around the lower pole of one adrenal in each case there was a hæmorrhage in the retroperitoneal fat. Microscopical examination proved that the dark masses in the lungs were infarcts. The small vessels in the internal organs and the retroperitoneal fat were congested, but the skin and subcutaneous fat were practically bloodless, and the spleen appeared to be less congested than the other organs. In various organs the blood vessels, ranging from capillary size to a diameter of about 100 μ, showed striking changes. The red blood cells were tightly packed in the vessels and some of them stained poorly. In a number of the smaller vessels there were eosinophilic hyaline masses which had shrunk away from the vessel walls. These were particularly obvious in the inner zone of the adrenal cortex. There were two small hæmorrhages into the lining of the stomach.

The author emphasizes that what happens to the tissues depends on the time they are exposed to any given temperature. If the critical time-temperature level is not exceeded the tissues become normal after return to normal surroundings; if it is exceeded, then local lesions occur or death results. If the chilling has been severe enough, the minute vessels show an increased permeability, plasma passes through the wall, and the red cells are left tightly packed in the vessel, forming a mechanical obstruction to further flow of blood. This in turn produces anoxæmia and tissue damage. If the patient survives, various local lesions—such as degeneration of the cells in the vessel walls, foci of cellular and intercellular œdema in the skin, degeneration of the sweat glands, muscle necrosis, and degeneration of nerves—result from the circulatory upset.

Experimental work on the cause of death from exposure is reviewed. Death is thought to be due to decreased dissociation of oxygen at low temperatures and diminished capacity of the tissues for utilizing oxygen. Other suggestions made are that death may be due to shock, damage to the central nervous system, or the direct effect of cold on the heart. It is suggested that cold alone may not be a sufficient explanation, but that additional factors—such as humidity, wind, snow, old age, hunger, disease, injury, excessive consumption of alcohol, lowered morale, and exhaustion—may be important.

Gilbert Forbes.

Recent Observations on Chronic Changes in Joints of Divers due to Compressed Air. (Neuere Beobachtungen über chronische Gelenkveränderungen bei Tauchern durch Druckluftein wirkung.) HERGET, R. (1948). *Arch. Klin. Chir.*, **261**, 330.

The author examined 47 divers, aged from 22 to 72, who had followed their calling for up to 42 years and who had worked at depths of up to 193 ft. for periods of up to 35 minutes. Fourteen of them had had an attack of caisson disease before examination; in 10 there was radiological evidence of chronic changes in the shoulder-joints, and in 1 case in both hips as well. Three other patients who had not suffered from an acute attack also had changes in the shoulders. [A fine series of *x*-ray films shows the early peripheral increase in bone density in the head of the humerus, the consequent mottling and early irregularity of the outline of the head, and the late gross deformities.] In the hip-joints of the one patient in whom they were affected late changes were seen of a type differing from "ordinary" osteo-arthritis. Minute embolism followed by avascular necrosis, sclerosis, and degenerative changes constitute a clearly defined chain of events leading to a type of osteo-arthritis. Although the hip is most commonly affected in caisson workers the shoulder is more often affected in divers.

L. Michaelis.

Systematic Study of 100 Workers Using Pneumatic Tools. (Examen systématique de cent ouvriers se servant des outils pneumatiques dans les travaux publics.) DESPLAS, B., RAYMOND, V., and BERNARD, P. (1948). *Arch. Mal. prof.*, **9**, 388.

Investigations were made in the building trades on 101 workmen who used pneumatic tools varying in weight from 12 to 45 kg. with striking frequencies between 1,500 and 3,000 per minute. Most of them were used vertically. The ages of the men ranged from 23 to 66 years with an average of 43 years. Some had worked for only a few weeks, others for 40 years; most had worked for 10 or more years. As a rule 2 men worked with each tool during an 8-hour day, sharing the work between them. Most of the men were well developed; about a dozen made spontaneous complaint of various pains and many others did so when questioned. Seven men suffered from finger pallor, and stiff or crepitant joints were found in 3 others. Of the 101 workers 16 were examined in detail. Radiographs showed various patches of decalcification in bone, usually in the carpus. Some men had exostoses or intra-articular and more commonly extra-articular foreign bodies. Some epiphyses were malformed.

Vacuolation of bone was found in 40% of cases. The os magnum was affected most frequently, then the semilunar and the scaphoid. Many of the changes seen in the os magnum are thought to have followed old undiagnosed fractures. Attention is drawn to Böhler's view that Kienböck's disease of the semilunar is occupational and occurs in those who make sudden and repeated dorsiflexion of the wrist. Ossifications of tendons were

sometimes noted, generally round the elbow. No reference is made to vascular disease, and the authors conclude that most of the workers observed may safely continue their usual tasks. *G. C. Pether.*

Interval of Useful Consciousness at Various Altitudes. HALL, F. G. (1949). *J. appl. Physiol.*, **1**, 490.

In an investigation into the effects of high altitudes and resulting anoxia on mental faculties, experiments were carried out on young healthy men ; a low-pressure chamber was used and altimeters to indicate the equivalent altitude. The subjects carried out tests involving simple mechanical responses to external visual stimuli. At a simulated altitude of 35,000 ft. the subjects responded to the signals correctly for a time averaging one minute, after which loss of consciousness followed in a few seconds. Similar results were obtained for altitudes between 35,000 and 42,000 ft.

The time during which the subject could successfully perform the tests was lengthened by blood transfusion and shortened by previous removal of one litre of blood. The transfusions raised the oxygen capacity of the subjects from an average level of 15·6 to one of 17 g. hæmoglobin per 100 ml. blood, and the blood-letting lowered the oxygen capacity from an average level of 15·3 to one of 13·3 g. hæmoglobin per 100 ml. blood. The author considers that the experiments suggest a possible means of increasing tolerance to altitude. *R. P. Foggie.*

The Effect of Exercise on Renal Plasma Flow in Normal Male Subjects. CHAPMAN, C. B., HENSCHEL, A., MINCKLER, J., FORSGREN, A., and KEYS, A. (1948). *J. clin. Invest.*, **27**, 639.

The effect of exercise on renal plasma flow was studied in patients with moderate water diuresis who emptied the bladder by voluntary micturition. The authors estimated *para*-aminohippurate clearances in 9 healthy male subjects, aged 21 to 32 years, before, during, and after exercise on a motor-driven treadmill. Clearance values were corrected for surface area. Of 59 experiments with a total of 140 clearance periods, 35 were acceptable as having two or more basal values within 10% of each other. In the large series the basal corrected mean for consecutive determinations in triplicate was 613±107 ml. per minute. In all cases there was a progressive decline in renal plasma flow as work proceeded. After the subjects had walked for 32 minutes at 3 miles per hour at zero gradient, 3 miles per hour at 5% gradient, and 3·5 miles per hour at 10% gradient, the renal plasma flow was decreased by an average of 15, 27, and 35% of resting values. Recovery of renal plasma flow to basal levels was incomplete 40 minutes after the exercise and was considerably slower than that of the pulse and blood pressure. If this decrease in renal plasma flow represents a diversion of blood to working muscles, then from these results, during the lightest work noted above, 150 ml., and for the heaviest work 330 ml., of whole blood per minute are diverted. *J. Maclean Smith.*

Estimations of the Decrease in Effective Blood Volume when Pressure Breathing at Sea Level. HENRY, J. P., HENDRICKSON, I., MOVITT, E., and MEEHAN, J. P. (1948). *J. clin. Invest.*, **27**, 700.

Recent studies of the effects of breathing air at increased pressures (Barach, *et. al.*, *J. Aviat. Med.*, 1947, **18**, 73), have suggested that the tendency towards circulatory collapse which occurs is due to a decrease in circulating blood volume brought about by pooling of blood in congested limbs and loss of fluid into the tissues by increased filtration. In the present experiments the extent of fluid loss in normal subjects during pressure breathing was followed by determinations of hæmatocrit, hæmoglobin, and plasma protein values. Breathing air at 30 mm. Hg at sea level for 30 minutes caused a calculated loss into the tissues of 4% of the blood volume. Raising the pressure to 53 mm. Hg doubled the fluid loss. Wearing a pressure jacket greatly increased comfort but did not decrease fluid loss. Counter-pressure on the legs decreased fluid loss by about 20%. *R. A. Gregory.*

Effect of Muscular Exercise on Dark Adaptation. WENDLAND, J. P. (1948). *Amer. J. Ophthal.*, **31**, 1429.

It is stated that heavy exercise causes a definite decrease in the ability to see at night.

Physiologic Effects of Industrial Solvents. FOULGER, J. H. (1949). *J. Amer. med. Ass.*, **139**, 826.

INDUSTRIAL LUNG DISEASE

Employment of Persons with Pulmonary Tuberculous Lesions. JOCZ, M. W., PRENDERGAST, J. J., and BIRKELO, C. C. (1948). *Occup. Med.*, **5**, 496.

Between 1942 and 1946 a group of physically " substandard " workers was studied by the medical organization of an engineering firm. Only those capable of full-time work were accepted, and no special recommendations as to job were made, except to forbid exposure to silica. The chests of all new applicants for jobs were radiographed as a routine, but old employees were radiographed only if there were definite indications. At 241,000 examinations made in one area active pulmonary lesions were found in 661 people, and these were considered unemployable ; 1,757 others with inactive lesions were accepted for employment or re-employment. Most of those with active lesions were fresh applicants and were not aware of their disease. The greatest number of tuberculous applicants was seen in 1943, because most healthy men had entered the armed forces ; some men had obviously left sanatoria against advice in order to do war work.

Of all those with tuberculosis, 8·9% had had sanatorium treatment ; nevertheless, one-fourth of them had active lesions and were unemployable. The authors were impressed by the staying power of 21 workers with thoracoplasties and inactive lesions. During the period of study 173 people out of the 241,000 developed active lesions ; 39 had known lesions which were considered inactive at the outset ; in 51 the films had been normal ; 83 had developed lesions but had had no initial radiograph. The 39 with known lesions worked for an average of 21·8 months before activity was discovered. A rate of 1·25% for activation of inactive lesions is suggested. The 51 originally considered normal worked for an average of 28·2 months before the disease developed. In the 5-year period 47 individuals with active lesions were able to return to work after adequate therapy lasting for an average of 15 months. There was no unusual concentration of active disease in any department. The authors conclude that the employment of persons with certain types of inactive pulmonary disease is practicable, that such employment in a highly mechanized industry does not harm these persons or their fellow workers, and that the work record of tuberculous

employees is as good as that of other substandard employees. [No evidence is given to support the last conclusion.] *J. N. Agate.*

Studies on Aerosols V. Effect of Dust and Pneumodilating Aerosols on Lung Volume and Type of Respiration in Man. DAUTREBANDE, L., ALFORD, W. C., HIGHMAN, B., DOWNING, R., and WEAVER, F. L. (1948). *J. appl. Physiol.*, **1**, 339.

The authors investigated the effects on 3 healthy human subjects of the dusts of: aluminium, aluminium hydrate, willemite, dolomite, calschist, copper sulphide, and zinc sulphide. The mean size of the dust particles was less than 1 μ, and the authors studied the effects of pneumodilating aerosols on pulmonary volume and type of respiration before and after the inhalation of the dusts.

All the dusts produced prompt reduction in pulmonary volume, nor was there any detectable difference between aluminium dusts (which are used therapeutically) and the others. The authors consider that, since the reduction in volume often exceeded 1 litre, the smaller respiratory passages were probably involved in the constriction. Tachypnœa accompanied the reduction in pulmonary volume. Repeated administration of dusts reduced pulmonary volume still more. Pneumodilating aerosols counteracted the reduction in pulmonary volume when given after the dusts; they prevented bronchoconstriction if given before the dust. The aerosols used included 0·2% "aleudrin," a mixture of aleudrin and neosynephrine (0·1 and 0·25%), this combination being found more effective than either drug alone, and procaine 0·1 to 0·2% in a mixture with tincture of lobelia, 0·5 to 1%.

The dusts produced no subjective irritation in the 3 subjects used.

In the treatment of silicosis aluminium dusts may be used, but as these produce bronchoconstriction they may not reach the depths of the pulmonary tree. The workman so treated, returning to work involving greater respiratory efforts, will draw harmful dusts more deeply into his lungs than the therapeutic dusts. The authors suggest that the aluminium dusts would be more deeply distributed if pneumodilating aerosols were used before inhalation of therapeutic dusts. Furthermore, pneumodilating aerosols might be profitably administered to "wheezy" subjects at the close of a working day, to relieve them of their respiratory distress.

A. T. Macqueen.

Evaluation of Several Methods of Functional Examination of Respiration in the Diagnosis of Silicosis. (In Russian.) EVGENOVA, M. V. (1948). *Klin. Med., Mosk.*, **26**, No. 10, 43.

An investigation was made into the functional capacity of the lungs in workers exposed to dust and workers whose lungs showed evidence of silicosis. In addition to the usual functional tests the following were measured: vital capacity, tidal, complementary, and reserve air (Krogh spirometer), pulmonary ventilation per minute, length of time for which deepest inspiration could be maintained, reaction of pulse and respiration rate to graded exercise, and oxygen debt after graded exercise.

Vital capacity was measured in 431 cases including 98 controls. The results in men and women were the same. The control group had a mean vital capacity of 3,686 ml. With increasing severity of silicosis the vital capacity fell from 3,695 ml. in stage 1 to 2,190 ml. in stage 3 of the disease. The initial increase in the vital capacity in stage 1 compared with the control group was thought to be due to the development of a compensatory emphysema in the initial stages of the disease. There was a fall in the tidal air only in stage 3; the reduction in vital capacity was mainly due to a fall in the complementary air and to a lesser extent to a fall in the reserve air.

In 51 controls and 164 cases of silicosis pulmonary ventilation rate per minute was studied. A steady rise was observed in the cases of silicosis compared with normal subjects. Graded exercises led to a normal response (pulse and respiration rate returned to normal within 3 minutes) in 84% of the healthy control group (253 cases) whereas in the silicosis group (705 cases) in stage 3 there was a normal response only in 3%. Oxygen debt after graded exercise was investigated in a small group of people; no definite conclusions were reached. It is suggested that some of the functional investigations may be of value in the diagnosis of an early silicotic process in the lungs. *N. Chatelain.*

Some Changes in the Blood in Silicosis. (In Russian.) GEUKIN, S. M., GELFON, I., MIGINA, N., RASHEVSKAYA, A., and SHILOVA, A. (1948). *Klin. Med., Mosk.*, **26**, No. 10, 49.

The authors found that certain blood changes may occur early in silicosis. The raised erythrocyte sedimentation rate found in a high percentage of cases of early silicosis is explained by the presence of secondary infection, bronchitis, pleurisy, and slight toxæmia which are known to occur, even in the early stages.

The viscosity of the blood was studied in 249 cases—31 healthy controls, 103 subjects exposed to dust but without signs of silicosis, and 59 with stage-4 silicosis, 29 with stage-2 silicosis, and 27 with stage-3 silicosis. The viscosity of the blood was over 4 in 45% of controls, in 37% of those exposed to dust, in 50% of stage-1 cases, in 70% of stage-2 cases, and in 91% of stage-3 cases. In order to eliminate the possibility that tuberculosis had increased the viscosity of the blood the same investigations were carried out on a group of patients free from tuberculosis but with silicosis; the same results were obtained. When investigating the viscosity of the blood in relation to the hæmoglobin value the quotient of Hesse used was $\frac{\text{(hæmoglobin percentage)}}{\text{viscosity of the blood}}$. It was found that even in early cases of silicosis the quotient was lower than 17·8 (normal). The rise in the blood viscosity was thought to be due to a rise in the mean corpuscular volume of the erythrocytes of up to 92·2 μ^3, found to occur in a group of 160 cases of silicosis. The possible causes of a rise in the mean corpuscular volume are discussed.

Experiments *in vitro* showed that the addition of silicon dioxide to whole blood raised the viscosity of the blood. *N. Chatelain.*

Asbestosis: VI. Analysis of Forty Necropsied Cases. LYNCH, K. M., and CANNON, W. M. (1948). *Dis. Chest*, **14**, 874.

The authors report an analysis of 40 cases in which asbestosis was found at necropsy. Cases are divided into minor, medium, and advanced grades, the advance of the disease and the prominence of asbestosis bodies showing a direct relation to the duration of exposure. In 4 of 14 cases of advanced grade, pulmonary tuberculosis was the primary cause of death. Only in cases of recent exposure was recent fibrosis found, this providing support for Gardner's belief that fibrosis does not progress indefinitely after cessation of exposure (*J. Amer. med. Ass.*, 1938, **111**, 1925).

Presence of asbestosis bodies is not invariable in experimentally-produced asbestosis but is more typical of human asbestosis. Lung damage occurs before the formation of asbestosis bodies, which may represent an attempt by the tissues to segregate the particles; they persist in the lung indefinitely. Pleural fibrosis is an outstanding, though not invariable, feature of asbestosis; the reason for this is not clear, as the particles do not reach the pleura.

The occurrence in asbestosis of nodular fibrosis of silicotic type, which King *et al.* have produced experimentally (*Thorax*, 1946, **1**, 188), was confirmed in 8 cases. Carcinoma of the lung occurred in 3 out of 40 cases—an incidence of 7·5%, which is 7 times greater than general incidence—all in cases of medium- or advanced-grade asbestosis. This figure is in accordance with the experience of other workers, and calls for further study. Tuberculosis occurred in 6 cases, but it is suggested that this incidence is not significant; this is not quite convincing. Gardner stated that experimental asbestosis does not predispose to tuberculosis.

[Unfortunately the occupational histories of these cases are often incomplete.] *L. W. Hale.*

SKIN DISEASES

Chronic Progressive Infectious Gangrene of the Skin. A Patient with Exposure to Cold Wave Solution. SIMEONE, F. A., and HARDY, H. L. (1948). *Ann. Surg.*, **128**, 1112.

A case of chronic progressive gangrene of the skin of the abdominal wall is described. As the infection was accompanied by leucopenia, and the patient was a hairdresser, it is suggested that thioglycolic acid, an ingredient of "cold-wave" solution, was responsible. The patient, aged 33, had for the past 2 years shown signs of unusual susceptibility to infection. The gangrenous area, which eventually involved the whole right lower quadrant of the abdominal wall, started in two operation scars. A high temperature, a high pulse rate, and leucopenia were observed. Investigations for syphilis, actinomycosis, brucellosis, and amœbiasis were negative. There was no response to sulphonamides or penicillin, or to activated zinc peroxide. The whole affected skin was removed and new skin grafted; the patient was cured. *F. B. Cockett.*

Granulomatous Moniliasis Resembling Blastomycosis (Gilchrist's Disease). MOORE, M. (1948). *Mycopathologia, Amst.*, **4**, 272.

A granulomatous lesion with the clinical characteristics of blastomycosis arose on the back of the hand of a farmer aged 56 in the site of an injury from a barbed-wire fence. *Staphylococcus aureus* and *Candida albicans* were cultured, but organisms were demonstrated with difficulty in stained sections of tissue, where they were seen as yeast-like bodies in large macrophage cells and stained with methylene blue. It was not possible to demonstrate blastomyces. The lesion was a granulomatous mass with a reddish, thickened border from which beads of pus could be expressed. Intraperitoneal inoculation of a subculture into a mouse produced visceral abscesses of similar character from which the fungus was recovered. A variety of treatments over 6 months failed to effect a cure, though some healing occurred in the centre of the lesion. *John T. Ingram.*

Causative Diagnosis of Contact Dermatitis. II. A Classification of Patterns on the Hands. WALDBOTT, G. L., and SHEA, J. J. (1948). *Arch. Derm. Syph., Chicago*, **57**, 975.

A classification of contact dermatitis of the hands is worked out from the characteristic patterns made by objects in contact with the palmar aspect of the hands. Clearly defined patterns are only rarely obtained in contact dermatitis of the dorsum of the hands. The pattern varies with the shape of the hand, strength of grip, and size and consistency of the object. The classification includes such varieties as the finger-tip type due to touching plastic knobs or buttons, the finger and palm types due to grasping objects such as ropes, golf clubs, bicycles, or car steering wheels, and the palm types due to holding objects in the palm of the hand. *G. A. Hodgson.*

Contact Dermatitis of Celery Farmers. WISWELL, J. G., IRWIN, J. W., GUBA, E. F., RACKEMANN, F. M., and NERI, L. L. (1948). *J. Allergy*, **19**, 396.

Examples of Cross Sensitization in Allergic Eczematous Dermatitis. BAER, R. L. (1948). *Arch. Derm. Syph., Chicago*, **58**, 276.

Textile Dermatitis in Men. PETERKIN, G. A. G. (1948). *Arch. Derm. Syph., Chicago*, **58**, 249.

Dermatitis of the Hands due to Ingested Allergens. WINSTON, B. H., and SUTTON, R. L. (1948). *Arch. Derm. Syph., Chicago*, **58**, 335.

ACCIDENTS AND ORTHOPÆDIC SURGERY

Hydrofluoric Acid Burns. (Flussyreætsninger.) HANSEN, G. A. (1948). *Nord. Med.*, **39**, 1610.

Hydrofluoric acid destroys all living tissue with which it comes into contact, its effects varying with the concentration and duration of contact of the acid and the thickness of the epidermis. Solutions under 60% act only after a latent period and 10% solutions if quickly and thoroughly washed off may do no damage, but even 1% solutions over a long enough period will lead to deep necrosis, sparing, however, the stratum corneum. The vapour from 2 to 3% solutions irritates the eyes and if inhaled may produce temporary and occasionally permanent anosmia. Strong solutions splashed on the skin produce excessively painful necrosis of tissues down to and including bone; even when treated adequately the lesions may take months to heal. The epidermis may show only bullæ, but usually a thick, tough, yellow coagulum of epidermis and corium forms, under and around which there is an area of black gangrene with a reddish edge. Pain, sometimes not controllable with morphine, is present when the corium is involved and colliquative necrosis may go on for days.

Adequate, and prompt, treatment is essential. Prophylactic measures for workers include wearing of rubber gloves, boots, and aprons, and good ventilation of rooms. Thorough washing with weak alkali is advocated, assisted by the application of ice-cold alcohol to constrict the lymphatics. Eyes should be washed out with saline solution, and 3% sodium thiosulphate drops should be instilled every 5 minutes. The essential part of the treatment, however, is the injection of 10% calcium gluconate into, around, and deep to, the affected area.

[The volume required is not stated, but 40 ml. was used for a hand.] Equal parts of magnesium sulphate and glycerin are massaged in as a salve at once and fresh compresses are afterwards applied twice daily. This method gives good results if employed in the first 8 hours and is helpful up to 4 days after injury. The calcium rapidly controls the spread of necrosis and stops the pain almost at once. With immediate treatment burns heal in about 6 weeks ; with a few hours' delay healing may take months. In cases of eye burns, as well as instillation of cocaine and atropine, injections subconjunctivally of 1% calcium gluconate may be necessary.

A. M. M. Wilson.

GENERAL

Job Performance of Physically Impaired Persons in Industry. FELTON, J. S. (1948). *Occup. Med.*, **5**, 466.

The Oak Ridge National Laboratory employed 452 (11·1%) people with physical limitations. The performance of 300 of these is compared with that of 300 normal controls. The disabled men had all manner of abnormalities : 32·6% had hernia. An assessment of emotional adjustment, based on the answers given to significant items in a questionnaire consisting of 200 queries (Cornell Index), was made for comparison. Accident rates were also compared. The average scores of the two groups did not differ as regards punctuality, attendance, or attitude, but the quantity of work done appeared to be greater in the " impaired " group, despite the variety of jobs, while scores for ability and quality of work were rather better in the unimpaired. The impaired workers had a slightly greater tendency to neurosis. The physically normal group had a significantly higher accident rate, but the number of injuries per worker was the same in both groups. The total scores suggest no significant difference between the job performance of the normal and impaired groups, nor between the larger subgroups of the impaired, for example, those with cardiovascular disease, hernia, or tuberculosis. There was a low correlation between the Cornell Index scores and the supervisors' ratings ; the former gives little indication whether or not a man is adjusted satisfactorily to his job.

The author concludes that these performances reveal the fallacy of applying rigid physical standards in industry, and that disabled persons are valuable, productive, and safe employees. [Physical impairment and disability are confused. The former was determined at pre-employment examination, and all varieties seem to have been included, regardless of whether a worker was handicapped in the job he was given. Measurements of job performance take no account of individual workers' problems. Many workers left before their performance could be studied.] *J. N. Agate.*

The Incidence and Treatment of Infective Ear Disease in Factory Employees. JOHNSTON, C. M. (1948). *Brit. med. J.*, **2**, 1049.

In a random sample, 1,902 workers in Birmingham factories were examined by the author, an otologist ; in 2·6% there was otitis externa, in 1·6% dry perforation of the tympanic membrane, and in 3·3% active suppurative otitis media.

A factory examination and treatment service was established and the author records the experiences in a group of factories employing 51,900 persons. Some 389 patients with otitis externa presented themselves : in the acute diffuse variety application of 0·5% gentian violet in 50% spirit, after scrupulous cleansing of the meatus, was the treatment of choice. In chronic otitis externa, moist conditions were treated with a similar gentian violet preparation ; dry conditions by oily preparations. Infection recurred in 20% of cases of furunculosis and chronic otitis externa. The variety of otitis externa with granulations on the surface of the tympanic membrane or meatal walls is discussed.

With chronic suppurative otitis media 274 patients attended and received treatment ; granulations or polypi (present in 28·8% of cases) were treated by removal and application of trichloroacetic or chromic acid ; epithelial debris or cholesteatoma (the last diagnosed in only 6 of the 274 patients) was removed by forceps or probe and syringing of the attic. Routine daily treatment consisted in removal of pus by syringing, followed by instillation of 50% spirit or light dusting with a powder, usually of sulphanilamide and boric acid in equal parts. Treatment of any infection in the nose, mouth, or pharynx causing spread through the Eustachian tube was regarded as essential. Of 213 ears completely treated there was " immediate cure " in 81%, the average duration of treatment being 5·2 weeks (limits 1 week to 7 months). Examined by the otologist 6 to 18 months later 17 of 37 such cases of " immediate cure " showed relapse.

T. A. Clarke.

Infected Hands Treated with Systemic Penicillin. BARCLAY, G. A. (1949). *Brit. med. J.*, **1**, 175.

This article describes the treatment of 380 cases of infected hands with systemic penicillin. It is difficult to summarize, but the following are the main conclusions reached. A single daily dose of 200,000 units was given, which in severe cases was raised to 300,000 units. The present tendency would be to give 300,000 units, preferably of the procaine preparations. Penicillin cream was used locally in superficial infections. Saline baths were not employed, and physiotherapy usually proved unnecessary. Cases of paronychia and terminal pulp infection were compared as far as possible with similar cases treated before the introduction of penicillin. In both groups the rate of healing was accelerated and complications were fewer.

Twenty-seven cases of terminal pulp infection were treated conservatively. Five infections resolved without surgery ; the remainder had to be operated on, but a delay in operation did not result in complications and healing was slightly more rapid than average. [This is an important observation as in the past the teaching has been that pulp infections should be treated by immediate surgery for fear of bone necrosis. It now seems that in certain cases the condition will subside without surgery and that delay in active treatment will not endanger the underlying phalanx.] Four cases of acute tenosynovitis were treated, all with perfect results. In one of these the infection subsided without operation. [Such cases are rare but important. In the past the results were uniformly bad.] Admissions to hospital of cases of infected hands fell by over 50% despite the fact that attendances at the clinic rose considerably in the period under discussion.

Except in cases of paronychia, penicillin-sensitive organisms were isolated in every case save one, in which penicillin-resistant staphylococci were found. In 15 of 94 cases of paronychia there were penicillin-resistant organisms, and in these cases healing took slightly longer than in the others.

The main conclusion is that systemic penicillin is of considerable value in the treatment of infected hands because it greatly diminishes the incidence of severe infections, decreases the period of disability, and cures the more serious types of infection with a perfect functional result. *E. C. B. Butler.*

Infections of the Hand. LOUDON, J. B., MINIERO, J. D., and SCOTT, J. C. (1948). *J. Bone Jt. Surg.*, **30B**, 409.

In 2 years 723 patients at Oxford lost the equivalent of 5 months' work for 100 men from infections of the hand. The results of a method of treatment used during the last 6 months in 69 consecutive unselected cases are analysed. Instead of incision and drainage, excision and suture were carried out wherever possible, with full use of penicillin.

Cases are classified according to the site of infection and in four degrees of severity from throbbing and tenderness to discharge. In only half the cases was there a history of trauma. Bacteriological examination shows that pure *Staphylococcus aureus* was responsible in 87%; in the remaining cases *Staph. aureus* and β-hæmolytic streptococci were grown or culture was sterile. Most organisms were penicillin-sensitive. Treatment consisted of immobilization, elevation, rest in bed where possible, and administration of 50,000 units penicillin 3-hourly with vitamins A, C, and D. Operation was performed usually within 24 hours of admission under general anæsthesia with full aseptic precautions and application of a tourniquet. Incisions follow the line of skin creases, the infected area is exposed, and necrotic tissue is excised. After light dusting with penicillin powder the incision is closed by interrupted skin sutures. Where necrosis of skin has occurred a graft is used to effect closure. In the early stages of tendon sheath infection the sheath is irrigated with penicillin solution after opening, but later infections are treated by excision of necrotic tissue. After operation immobilization in plaster is continued until healing has occurred. Results show that of the 69 lesions 38 without skin necrosis healed in an average time of 7·3 days while the other 31 with some degree of skin loss healed in 19·6 days. *G. Blundell Jones.*

Dental Conditions in Workers Chronically Exposed to Dilute and Anhydrous Hydrofluoric Acid. DALE, P. P., and MCCAULEY, H. B. (1948). *J. Amer. dent. Ass.*, **37**, 131.

Analyses of data collected on 35 men chronically exposed for from 2 to 33 years to hydrofluoric acid fumes in a production plant, and on 11 non-exposed office and warehouse workers, revealed a lowered incidence of caries as a result of exposure. Significant differences between exposed and non-exposed groups of individuals were obtained for the number of carious and filled teeth, number of fillings, and number of tooth surfaces involved by fillings and cavities. Active carious lesions among the exposed individuals were encountered infrequently.

Radiographs revealed a change in the trabecular pattern of the osseous structure of the maxilla and mandible in 13 of 15 workers exposed for 10 years or more, and in 10 of 20 employed for less than 10 years. The bone changes were characterized by an increase in the number and thickness of trabeculæ and a corresponding decrease in the intratrabecular or canalicular spaces. Sclerosis and general disease of bone were not encountered. However, more recent radiographs revealed in one case a marked radio-opacity in practically all the bones of the skeleton without subjective manifestations of fluoride toxicity.

The fluoride excretion of 34 workers in contact with hydrofluoric acid varied considerably, with a mean of approximately 10·78 (±9·3) mg. of fluoride per litre of urine. This contrasts with the fluoride output of 0·67 (±0·63) mg. of fluoride per litre for 7 non-exposed men. On the basis of this single urine analysis there was no apparent correlation between urinary fluoride excretion and age of the employee, years of exposure, or bone changes. *R. Robertson-Ritchie.*

Disturbances of Smell in Workers in the Perfume Industry. (Le turbe dell'odorato negli operai addetti nelle industrie dei profumi.) MANCIOLI, G. (1948). *Rass. Med. industr.*, **17**, 104.

The author describes anosmia as seen in perfume workers. The materials employed are leaves, flowers, and roots of plants, which yield oils and essences; certain animal products such as musk or ambergris, and synthetics of various kinds. The essential oils are often extracted with spirit or some other volatile solvent. Excipients are often used in order to retard evaporation of the scent. Colouring materials are added to some perfumes.

Headache, vertigo, neuralgia, sweating, vomiting, asthenia, or digestive disturbance may be encountered. Some disturbance is generally noted in workers engaged on extraction processes with ether or ammonium solutions, which are partly responsible for the symptoms though the matter extracted is likewise involved. Older operatives develop tolerances to many materials; Epistaxis and rhinitis, or swelling and inflammation of the eyelids, may develop. Dermatitis is seen, sometimes as an eczema, erythema, or papular or pustular eruption. Some oils, such as oil of geranium, act on the nervous system and may have a narcotic effect. Oil of aniseed causes excitation with spasmodic movements, fibrillary tremors, and irritability followed by drowsiness. The oils of lavender and rosemary have a similar effect, presumably from their content of terpenes. Irritation of the urinary tract has been attributed to some of the materials used in scent manufacture.

In the first section of the factory dentifrices were made, both in powders and pastes. Natural essences were used to give taste and aroma. In the second section bottles were filled with the base for ointments, and in the third beauty preparations were made. Marked hyposmia was observed in only a few of the workers in the first section. Loss of sense of smell was more frequent in the second and somewhat less common in the third. There appeared to be some direct relation between length of employment and loss of the olfactory sense. *G. C. Pether.*

Allergic Examination of Bakers and Millers. (In English.) CASTBERG, T., and SØRENSEN, C. M. (1948). *Acta allergol., Kbh.*, **1**, 283.

The frequency of diseases due to flour allergy was investigated among 130 bakers and millers. Allergic symptoms when working with flour were reported by 42. In 14 asthma was the main complaint, in 13 vasomotor rhinitis; 8 had eczema, 6 urticaria, and 1 hemicrania. When intradermal skin tests were made on 93 of the 130 bakers, 39 gave positive reactions to flour or flour-improving substances, though only 26 of these had allergic symptoms. Ammonia persulphate, on account of its tendency to cause eczema, is not used in Denmark as an " inprover." *A. W. Frankland.*

PROCEEDINGS OF THE ASSOCIATION OF INDUSTRIAL MEDICAL OFFICERS

FIFTY-FIFTH MEETING

The Fifty-Fifth Meeting of the Association was held on April 29 and 30 at the University College Hospital Medical School, Gower Street, London, W.C.1.

It was agreed that the annual subscription should be increased to two guineas and one guinea for Associate Members. The agreed alterations to the Constitution also included the provision that any Associate Member actively engaged in the practice of industrial medicine for twelve months might become an Ordinary Member at any time if he wished, by making application in writing to the Council. A further amendment provided for the admission to Associate Membership of qualified students attending courses for the Diploma in Industrial Health.

The clinical meeting was opened by Dr. Kenneth Harris, Senior Physician of University College Hospital, with a lecture entitled "The Rheumatic Heart as a Disability." Dr. Harris outlined the American classification of cardiac conditions under the four headings: ætiology; structure; function; complications. He described the Lewis classification, which is more widely used in this country and the main headings of which are: exercise tolerance; congestive cardiac failure; enlargement; valves; rhythm; ætiology; infection; vessels. Dr. Harris said that most patients asked three questions about cardiac lesions: "How did I get it? How badly damaged is it? How much can I do?" The last question was important in relation to employment. The exercise tolerance test was very useful; elaborate mechanical tests were not usually practicable, but a simple test such as stepping on to a chair twenty times gave valuable information provided that the normal limits of tolerance had been determined. The respiration and pulse rates should be measured before, immediately after, two minutes after, and four minutes after exercise (the last only if recovery had not been complete in two minutes). The limit of the patient's daily exercise should be just short of that which causes dyspnœa or pain. If there were evidence of congestive failure, or a recent history of failure, sedentary work should be advised.

Dr. Max Rosenheim, Assistant Physician to University College Hospital, spoke on the assessment of hypertension. Heredity and racial factors were important in the ætiology, but evidence was lacking as to the significance, if any, of such factors as the stress and strain of modern life. Psychological factors were important, and obesity appeared to be a predisposing factor. Dr. Rosenheim described the "cold-pressor" test for hypertension, in which early hypertensives showed a greater rise than normal in systolic blood pressure following the immersion of one hand in very cold water. People who tended to a nervous rise of blood pressure that settled on rest were more likely to develop hypertension later. There was in fact a "basal blood pressure" which could be estimated after rest in bed or after sedation, and it was people with high basal pressures who were liable to develop complications. Transient high rises of pressure occurred in people who might possibly develop hypertension later. The basal pressure was important in prognosis.

On April 30, Dr. Rosenheim spoke on the industrial aspects of coronary arterial disease. He thought the prognosis in cases of angina was surprisingly good; the average duration was seven to ten years. The first attack of coronary thrombosis was often fatal, and many who did not die were unable to return to work. If there were congestive failure or a marked lowering of the blood pressure, the patient would not again be fit for a full day's work. However, some patients lived ten years or more without recurrence of symptoms. In relation to work each patient had to be dealt with individually, the physical nature of the work and the emotional stress which the work produced in the patient being taken into account.

The meeting concluded with a talk by Dr. Kenneth Harris on the interpretation of the electrocardiograph.

BIRMINGHAM AND DISTRICT GROUP

Chairman: Dr. W. Jeaffreson Lloyd,
Messrs. Guest, Keen, and Nettlefolds Ltd.
Birmingham.

Hon. Secretary: Dr. J. G. Billington,
Messrs. G. E. C. Ltd., Witton, Birmingham.

A visit was paid by members of the Group to the Birmingham Corporation Salvage Department where they were shown round by the Chief Chemist, Mr. W. Weaver, B.Sc., F.R.I.C. This Salvage Department has been operating for many years and is a pioneer in the utilization of organic waste. Over 1,000 tons of refuse are collected each week at a cost of 22*s.* per ton and disposed of at a cost of 14*s.* per ton. This cost is offset by an income of £110,000 from the sale of fertilizers, feeding meal, etc. After being weighed the refuse is passed through totally enclosed screens to extract all fine dust (45 per cent. by weight), which is taken away to fill up low-lying land. Ferrous scrap metals are automatically removed by an electro-magnetic process and baled. Articles of salvage value are next removed manually from conveyor belts. Paper, cardboard, and textile waste are sorted and baled. Other items of salvage recovered include bones, non-ferrous metals, bottles, and jars. The remainder of the refuse is then incinerated, the heat generated being used for steam raising and generation of electricity for use in the plant.

The Group was impressed with the care with which the organic refuse, namely offal, vegetable refuse, condemned meat, and other waste from abattoirs and markets, is dehydrated, autoclaved, and made into pig and poultry meal and fertilizers. A special process of oxidation by chlorine of noxious gases is used to get rid of the di-methyl amides from the meat and the tri-methyl amides and hydrogen sulphide from fish. A production bonus scheme makes a heavy and dirty job more attractive and first-aid facilities receive careful attention.

LONDON GROUP

Chairman : Dr. G. E. Graves Peirce,
Medical Officer, Railway Executive,
Euston Station, London, N.W.1.

Hon. Secretary : Dr. R. E. W. Fisher,
Chief Medical Officer, South Eastern Gas Board,
Metropolitan Division, London.

A meeting was held in the London School of Hygiene and Tropical Medicine on May 19. Dr. Graves Peirce was in the Chair and Dr. Richard Doll read a paper on peptic ulcer and occupation. The literature provided evidence of a high incidence among fishermen. A survey of 6,047 workers had shown that duodenal ulcers are evenly distributed throughout the social classes but that gastric ulcer is predominantly a disease of the lower income groups. A significantly high incidence of duodenal ulcers was found among men in responsible positions in industry, and this was associated with a tendency to worry. As would be expected, agricultural workers did not suffer much from ulcers, but the incidence among drivers was close to that expected. No unusual incidence was found among shift or night workers, or workers forced to take meals at irregular hours. The incidence of ulcers varied greatly with age. The population between the ages of 15 and 64 was estimated to contain 5·8 per cent. of men with ulcers and 1·9 per cent. of women. Sickness absence due to peptic ulcer was estimated at thirty-two working days a year per 100 men. It was suggested that these figures demonstrated the need for supervision of diet in industrial canteens.

NORTH-WESTERN GROUP

Chairman : Dr. R. S. F. Schilling,
University of Manchester.

Hon. Secretary : Dr. G. Taylor,
A. V. Roe and Co. Ltd.,
Greengate, Middleton, Manchester.

A meeting of the Group was held at Manchester on Nov. 25, 1948, with Dr. R. S. F. Schilling in the Chair and eighteen members present. A paper was read by Prof. Robert Platt, Manchester Royal Infirmary, on hypertension. Prof. Platt said that normal blood pressure was difficult to define precisely. High blood pressure did not usually cause symptoms until the diastolic pressure was 110 or higher. There might even be no symptoms, with a very high diastolic pressure such as 150. The headache and dizziness frequently described by patients was due to the anxiety state induced by the discovery of the hypertension ; at all stages the diastolic pressure was a much more significant factor than the systolic.

Severe hypertension in young people was in 75 per cent. of cases associated with a renal lesion, especially chronic pyelonephritis ; hypertension was occasionally due to unilateral renal disease. It would be a wise precaution, therefore, that all patients, say under 45, should be fully investigated and that any patient in whom the history suggested the possibility of renal abnormality, or of urinary infection should have tests of renal function and an intravenous pyelogram.

It had to be borne in mind that essential hypertension was of two types. Benign hypertension was compatible with a life of fifteen to twenty years after its discovery, cardiac failure occurring at 60 to 65 years of age ; occasionally some patients had a hemiplegia. Malignant hypertension usually appeared at the age of 35 to 50, with concurrent renal and cardiac failure ; these cases had papillœdema and a high diastolic pressure, always over 140.

Heredity had been shown to be the most important known factor in essential, but not in renal, hypertension, and it operated in both benign and malignant cases ; so, in the absence of a family history, a renal cause should be suspected. Essential hypertension was an entity and not merely a label attached to undiagnosed cases. Pointers to the diagnosis of essential hypertension were a " middle-aged " appearance ; heredity ; a history of migraine ; lability of the blood pressure ; and arteriosclerotic changes. Usually the expectation of life in benign essential hypertension was not seriously reduced until the diastolic pressure was over 110—some would even say as high as 120. The fitness of people for ordinary work in middle age was therefore not seriously in question until the diastolic pressure was 110 or more.

A meeting of the Group was held on Feb. 24, 1949 in the Department of Occupational Health, University of Manchester. The Chairman welcomed members of the Association of Appointed Factory Doctors, and Dr. J. M. Davidson, Ministry of National Insurance, read a paper on the Industrial Injuries Act.

A meeting of the Group was held at Barrow-in-Furness on May 27. Twenty-three members, including many members from the Merseyside Group, were present. By the courtesy of Messrs. Vickers-Armstrong Limited, a full day's programme had been arranged. In the morning a visit was paid to the clinic in charge of Dr. J. Stuart, and to the recently added *x*-ray unit for fractures and serial chest examination. The manufacturing departments, with their multiplicity of operations, were also visited.

In the afternoon, Mr. A. Ronald, F.R.C.S., Orthopædic Surgeon, North Lonsdale Hospital, and Consultant Surgeon to the Works, demonstrated with a cine-radiographic film, movements of the joints of the upper and lower extremities ; he followed this with a talk on injuries to the hand and forearm.

The recently opened rehabilitation shop was a lofty, pleasant, well equipped room in which about twenty men were at present engaged in selected tasks.

The plate-rolling mills, the pre-fabrication units, and the stock-yards with several ships (varying from 35,000 tons to 8,000 tons) in various stages of construction were inspected.

SCOTTISH GROUP

Chairman : Dr. D. Gordon Robertson,
I.C.I. (Explosives Division), Nobel House,
Stevenston, Ayrshire.

Hon. Secretary : Dr. William Hunter,
William Collins Sons and Co., Ltd.,
144, Cathedral Street, Glasgow.

On March 9, an address was given to the Scottish Group by Dr. Alexander MacLean, Chief Supervising Tuberculosis Physician, Glasgow Area, on rehabilitation in respiratory disease. Dr. MacLean demonstrated types of cases which were dealt with by the unit, and arranged a display of occupational therapy. On April 13, a visit was paid to the Mines Rescue Station at Coatbridge, and on May 11, the Group went to the Shale Oil Refinery at Pumpherston, where Dr. J. Scott discussed dermatitis in that industry and reviewed methods in use for its prevention.

TEES-SIDE GROUP

Chairman: Dr. H. Fallows,
Dorman Long and Co., Ltd., Middlesbrough.

Hon. Secretary: Dr. J. B. Adamson,
Ministry of Fuel and Power,
Newcastle-upon-Tyne.

A meeting was held at Billingham on Feb. 23, when ten members heard a talk on hand injuries and the organization of a hand injury clinic, given jointly by Mr. David Brown, orthopædic surgeon, and Mr. T. G. Lowden, surgeon, Royal Infirmary, Sunderland. Mr. Brown said that the main causes of poor or delayed treatment of hand injuries were (1) poor diagnosis, and (2) lack of hospital organization to deal with this type of injury. A hand injury clinic should have an orthopædic surgeon and a general surgeon attached to it, and should be staffed by a team of surgeons of varying experience. The nature of the case would determine by whom it should be treated.

Mr. Brown described faults in technique which might lead to stiffness in a hand or fingers (one of the chief disabilities following injury), and he also discussed errors to be avoided in treatment. The supervision and management of patients following their discharge from hospital were discussed, and also the part to be played by the works medical officer in the treatment of patients.

Mr. Lowden said that, of 4,000 hand injury cases seen by him annually at the hospital out-patient department, approximately 25 per cent. were cases of septic hand. He emphasized the importance of early diagnosis and treatment in combating the possibility of permanent disability.

Various types of injury, burns, soft-tissue injuries, distal pulp infection, contused nail, etc., were described and methods of treatment were discussed.

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